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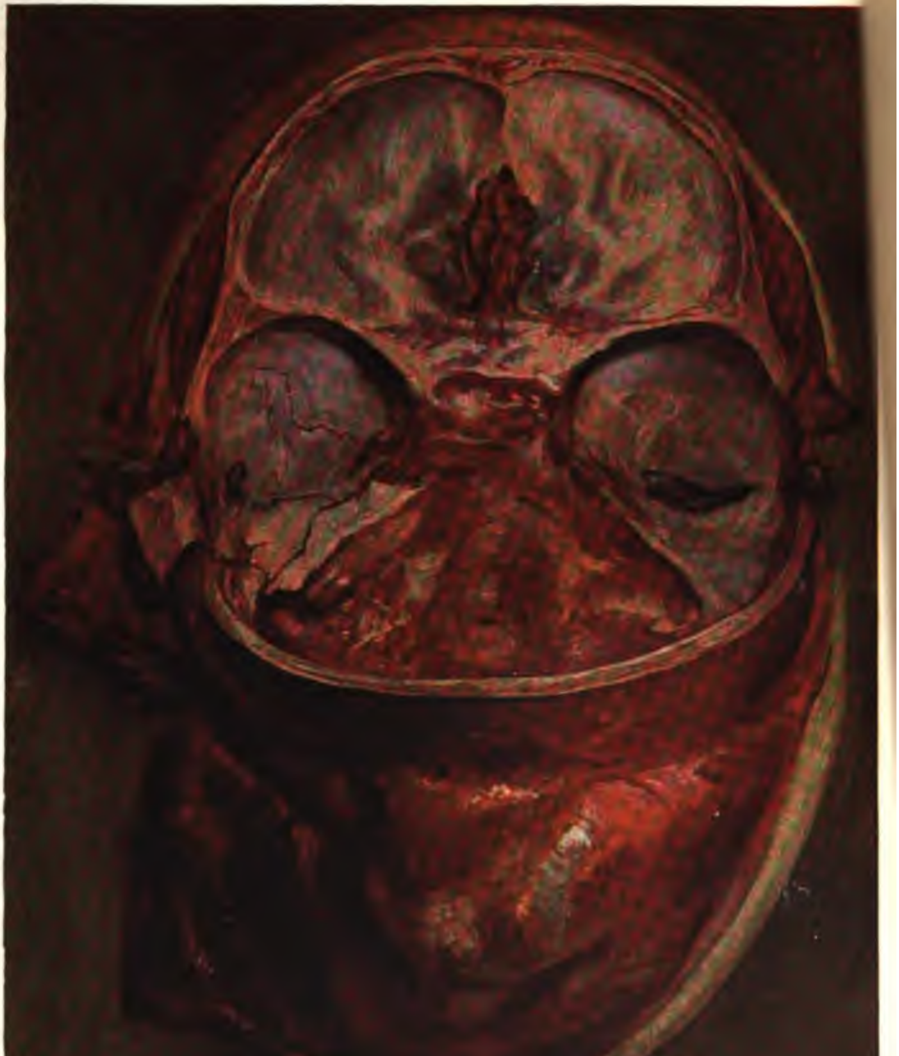
DIAGNOSIS AND TREATMENT OF BRAIN INJURIES



DIAGNOSIS AND TREATMENT OF BRAIN INJURIES

WITH AND WITHOUT A FRACTURE OF THE SKULL

BY

WILLIAM SHARPE, M.D.

PROFESSOR OF NEUROLOGIC SURGERY, NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL;
CONSULTING NEUROLOGIC SURGEON, MANHATTAN EYE AND EAR HOSPITAL, HOSPITAL
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AND NASSAU HOSPITAL, MINEOLA, LONG ISLAND, ETC., ETC.

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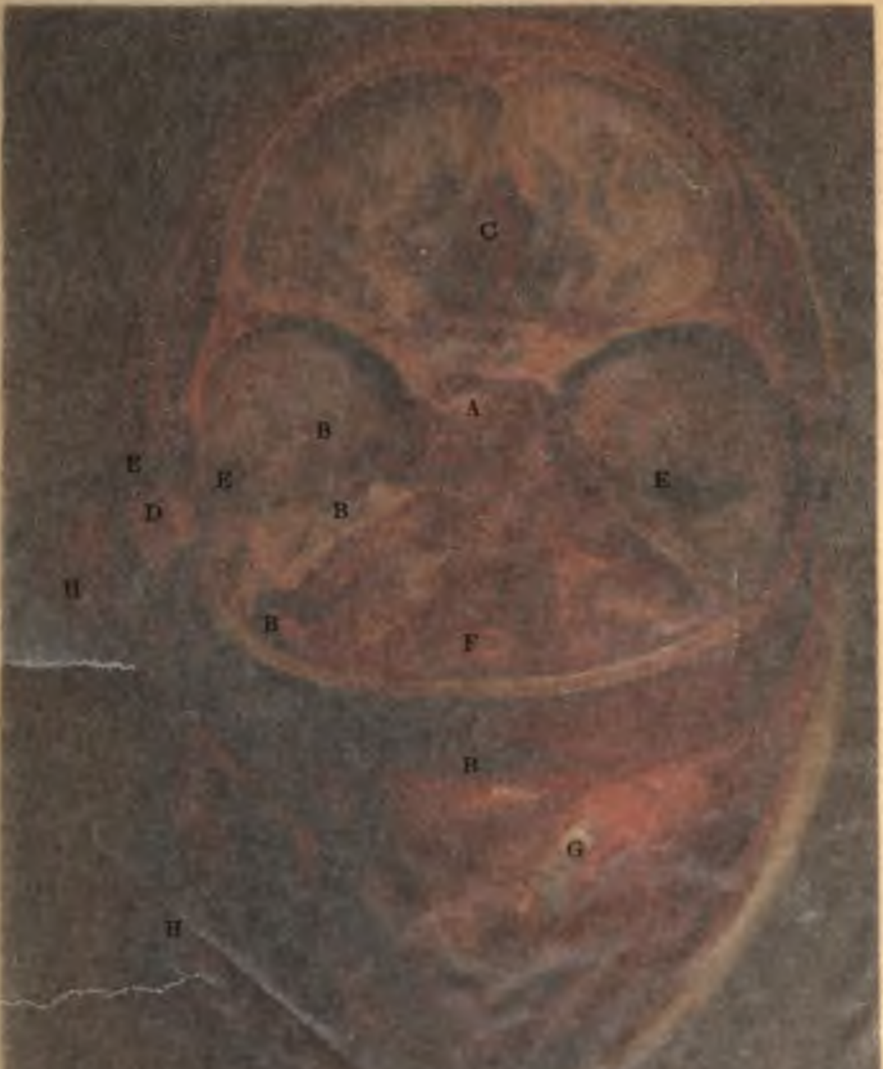

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E. RUBBER TISSUE DRAINS FOR LEFT AND RIGHT DECOMPRESSION OPENINGS
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G. REFLECTED SCALP
H. REFLECTED PORTIONS OF SCALP

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PREFACE

WITHIN the last few years, such an advance has been made in the diagnosis and treatment of brain injuries that I have considered it advisable and opportune to present in detail the recent developments of this subject—the more accurate methods of diagnosis and the descriptions and treatment of individual patients illustrative of the various types of brain injuries. I have used the designation "brain injuries" rather than "fractures of the skull," because in the treatment of these patients it is not so much a question of the presence or not of a fracture of the skull, but, of far greater importance, the presence or not of a brain injury, and, more important than any other factor in their treatment, the presence or not of an increased intracranial pressure, both as regards the immediate recovery of the patients and also their future mental and emotional status. Naturally, the treatment of the patient is not only concerned with the immediate recovery of life, but also the future condition of the patient—approximating and obtaining, if possible, a normal person after the injury.

To facilitate its presentation, this subject has been divided into the following main parts. The treatment is given in general, and then in detail, in each case, whether the expectant palliative or the operative treatment:

Part I. General Considerations: Pathology, Diagnosis, and Treatment; Operative Technic.
Part II. Brain Injuries in Adults. Illustrative Cases.
  1. Acute.
  2. Chronic.
Part III. Brain Injuries in Newborn Babies and Children. Illustrative Cases.
  1. Acute.
  2. Chronic.

A large number of individual cases has been presented in the belief that they will be of service to the general practitioner and to the general surgeon, and in the cases of brain injuries in newborn babies and children to the obstetrician and to the pediatrician. These patients are grouped according to the pathology and also to the method of treatment used; not only are the "successful cases" described in detail, but also the "poor results"—due in many instances to the injury itself, and then, only too frequently in the earlier patients of five years ago, to the time and method of treatment employed. These patients have all been followed carefully, and repeatedly examined; their present condition is reported in detail. Post-mortem examinations were made upon all of the reported patients who died. Those "accident" patients having brain injuries, however, were in the jurisdiction of the office of the coroner, and it was not always possible at times to receive the necessary permission—unlike the remainder of my neuro-surgical work, in which no operation is performed unless a written permission for an autopsy (if death should occur) is given *before* the

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operation is performed, and no operation is performed unless this written permission is given by the nearest relative. In those patients having acute brain injuries we were unusually fortunate, with few exceptions, in obtaining the necessary permission, and the data have been a source of much information to us in this work.

Intracranial hemorrhage, occurring in newborn babies as the result usually of difficult labor with and without the use of instruments, has been very much overlooked and its treatment neglected in the past, and it is only within the last five years that the chronic conditions, resulting from these intracranial hemorrhages, in the form of cerebral spastic paralysis, mental retardation and emotional instability with and without the serious complication of epilepsy, have been more commonly recognized. A number of these selected patients, both of the acute type at birth and of the chronic type later in life, are described in detail—the differential diagnosis considered and the pathology of the intracranial lesion demonstrated, either at operation or at autopsy.

The condition of post-traumatic neurosis is considered in a separate chapter; a number of illustrative cases are discussed in detail.

I wish to express my indebtedness to Doctor John A. Wyeth for the opportunity to make these studies, and from whom I have received numerous suggestions and valuable advice; his always helpful and constructive criticism has made this work possible; to my associates, Doctors Giles, Rochfort, Dunham, Lott and Espejo, for their careful, elaborate records and operative assistance; to Doctors Quimby and Welton for the excellent röntgenograms; to Doctors Hunt and Palermo for the important administration of the anesthesia; and to K. S. Gardner for the accurate drawings, photography, and the careful reading of the proofs.

W. S.

MAY, 1920.

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PART I

RECENT ADVANCES IN THE DIAGNOSIS AND TREATMENT OF BRAIN INJURIES

DIAGNOSIS AND TREATMENT OF BRAIN INJURIES

CHAPTER I

INTRODUCTION

THE mortality resulting from brain injuries is very high. During the period from 1900 to 1910, the mortality figures at three of the large hospitals in New York ranged from 48 to 68 per cent. of all cases of brain injuries. In a report[1] published in 1916 of the Cook County Hospital of Chicago regarding 1000 consecutive patients having "fractures of the skull," the mortality was 53 per cent. This death-rate is indeed appalling, and it undoubtedly accounts for the attitude of many doctors and of most hospitals toward patients having fractures of the skull, and particularly those of the base: if the patient recovers, remarkable—he had a fracture of the skull; if he dies—well, he had a fracture of the skull.[2]

It is this attitude of comparative hopelessness in the treatment of brain injuries that has allowed these patients to be almost neglected in the general hospitals. I well remember being severely reprimanded by my senior house surgeon for having admitted to the hospital from the ambulance a patient having "a fracture of the base of the skull," and not having sent the patient to Bellevue Hospital. As a house surgeon, my instructions were to keep the beds free of "fractures of the base of the skull,"—the reason being that so little surgically apparently could be done for these conditions; the patients either improved after a long convalescence, or more frequently they died—the treatment, other than the ordinary routine palliative procedures, being of little or no benefit.

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[1] Besley, F. A.: A Contribution to the Subject of Skull Fractures. *The Journal A. M. A.*, January 29, 1916, p. 345.

[2] Bailey, Pearce: Diseases of the Nervous System resulting from Accident and Injury, 1906, p. 86.

"In a study undertaken by me some years ago, an attempt was made to formulate some facts as to the fatality of brain injuries of a certain uniformity of type. Fracture of the base was chosen as the index, since it represents an extreme degree of violence and probably a similarity in the brain lesions. That this latter is true is shown by the greatest similarity in symptomatology, and also in conclusions as to mortality by different observers. Herr in 58 cases had 29 deaths, or a mortality of fifty per cent.; Van Nes in 82 cases had 39 deaths, or a mortality of forty-seven per cent.; Phelps, in 286 cases had 176 deaths, or a mortality of sixty-one per cent.; Dr. R. F. Weir and Dr. W. T. Bull, having kindly given me access to their histories at the Roosevelt Hospital, I found that 60 cases were received there during the years 1900 and 1901. By adding to these 9 cases of which I have private records, the resulting total is 69 cases. Of these 40 died, a mortality of fifty-eight per cent. Thus a fatal result is to be looked for in more than half of the cases of fracture at the base. The combined tables of Heer, Van Nes, Phelps and myself, comprising a total of 494 cases, show a mortality of fifty-seven per cent. In this connection it must be stated that some hospital records show a much higher mortality. In the great majority of cases death is due to the injury without intercurrent conditions."

DIAGNOSIS AND TREATMENT OF BRAIN INJURIES

The diagnosis and treatment of brain injuries have advanced most pidly within the last few years. The clinical symptoms and signs are so varied and frequently so confusing in these patients that it is a most fascinating field; apparently in many patients the more extensive the fracture of the skull, the less seriously is the brain injured; and, on the contrary, the most dangerous of brain injuries are frequently not even associated with a fracture of the skull. As is well known, the fracture in these patients (if we exclude depressed fractures of the vault which should always be elevated or removed) is possibly the most unimportant part to be considered in the treatment, whereas the presence of a marked increase of the intracranial pressure, with and without a fracture of the skull, should immediately cause the patient to be withdrawn from that large group of patients properly treated by the expectant palliative method, and the advisability of an early operative procedure to relieve the increased intracranial pressure should be considered.

In the treatment of brain injuries, with and without a fracture of the skull, if the patient is allowed to develop definite paralyses, a lowered pulse-rate, Cheyne-Stokes respiration and pulse, and that appalling group of extreme intracranial pressure signs, then I agree entirely with the opinion, so commonly now held that these patients "get along" just as well without operation as with operation at this late stage—the mortality being 50 per cent. and over; but patients with brain injuries should not be allowed to reach this dangerous stage of medullary compression, due to the high intracranial pressure; it should be anticipated by the accurate diagnostic methods now known, and if a marked increase of the intracranial pressure is ascertained, then an early relief of it should be advised, not only to save the life of the patient, but to lessen the post-traumatic conditions of changed personality, either of the excitable or of the depressed type, persistent headaches, early fatigue, occasionally epilepsy, and that long train of post-traumatic conditions in brain injuries, and due in the majority of patients to a prolonged increase of this intracranial pressure.

Acute Brain Injuries in Adults.—During the past six years (1913–1918) I examined and treated personally 487 adult patients having acute brain injuries, with and without a fracture of the skull; in only 155 of these 487 patients (that is, 31.8 per cent.) were there marked signs of an increased intracranial pressure, and therefore only these 155 patients were operated upon to relieve this increased pressure, whereas the remaining 332 patients did not show definite signs of an increased intracranial pressure, and were therefore treated by the expectant palliative method of absolute quiet, ice helmet and catharsis; if in shock, then the routine treatment of shock. It is thus seen that less than one-third of the patients having brain injuries, with and without a fracture of the skull, were operated upon, and approximately this same ratio has continued during the year (1919). It is this careful selection of patients, not only in regard to the advisability of an operation or not, and, if indicated, then the type of cranial operation used, but of the greatest importance—the ideal time for performing the operation—these factors have made it possible to lower the mortality of brain injuries from the average of 50 per cent. of most hospitals to 28.4 per cent. at the

Polyclinic Hospital, and if we exclude the non-operated moribund patients dying within three hours after admission to the hospital from shock, internal injuries, and in many cases the brain injury being but an incident in the patient's general condition, the mortality is lowered to only 17.9 per cent.

We now come to the most important and difficult question in the treatment of brain injuries, with and without a fracture of the skull: "If an operation is advisable, when should it be performed?" This question can more easily be answered by stating when the operation should *not* be performed; naturally, we must exclude the majority (about two-thirds) of brain injuries that do not have a definite increase of the intracranial pressure, and therefore no operation is indicated; the depressed fractures of the vault naturally should always be elevated or removed.

The two periods in which an operation is distinctly contra-indicated in cases of brain injury are, first, the condition of severe shock in the very beginning, and second, the condition of medullary edema and collapse—the death-knell of the patient. To advise a cranial operation upon a patient—no matter how badly the skull is fractured nor how extensive the intracranial hemorrhage seems, and that patient is in the condition of severe shock with a pulse-rate of 120 and higher, then the operation at that period takes away whatever chance the patient may have of surviving the shock; the operation is but an added shock, and merely hastens the exitus. No patient having a brain injury should be operated upon in this condition; the mortality is most high, and if the patient does recover from an operation in this period of extreme shock, then he recovers *in spite of* the operation. Cranial operations for brain injuries in this stage of shock were frequently performed in the past, and most disastrously, and thus operations were almost discredited in the treatment of brain injuries. The natural reaction following these early operations in the stage of severe shock was to wait until there could be no possible doubt that the patient was going to die, unless, as was thought, a cranial operation was performed—that is, the patient was permitted to reach the stage of medullary compression (a pulse-rate of 50 and below, irregular Cheyne-Stokes respiration and pulse) and profound unconsciousness before a cranial operation might be considered. This is a most dangerous stage for these patients to reach, and it is doubtful whether recovery can occur even with an operation at this late period, the mortality being very high. But if the patient has struggled through this period of medullary compression, and finally reaches the stage of medullary edema, when the pulse-rate begins to ascend quickly to 120 and higher, respirations become rapid and shallow—that is, the stage of medullary collapse—then we have the second period, when no patient should be operated upon; they all die, operation or no operation. I feel, therefore, that if these two extremes can be avoided, and the latter of these (medullary collapse) can certainly be anticipated in the operative treatment of brain injuries, and their signs cannot be overlooked, then the rational treatment of brain injuries depends upon the presence or not of a definite increase of the intracranial pressure, whether there is a fracture of the skull or not; in some of the most serious cases no fracture was present, either to be ascertained at operation in the operated patients or at autopsy. The aid of the en rays is important in the treatment of these traumatic cases only in nts with doubtful depressed fractures of the vault, and in latent ctures of the skull, where the bump is so apparently trivial that the patient might not be so carefully examined and treated as the condition would warrant; on the contrary, no patient with high intracranial pressure should be obliged to wait "overnight" or for a period of hours merely to secure a röntgenogram of the skull. It is of no importance in the treatment of these acute intracranial lesions whether a fracture of the skull is present or not. If there is a high intracranial pressure, as shown by the ophthalmoscopic examination and by the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer, then the cranial operation of subtemporal decompression is indicated to relieve this increased intracranial pressure, both by enlarging the intracranial cavity and by the drainage of possible hemorrhage and cerebrospinal fluid. It is not so much a question of removing the hemorrhage as it is of lessening the increased intracranial pressure; whether that pressure is due to hemorrhage or edema, the operative indication is the same. Many cases of cranial injuries at autopsy have revealed no hemorrhage at all—merely a " wet " edematous swollen brain, but sufficient to cause medullary compression, and eventually the death of the patient.

If an operation is considered advisable to relieve the increased intracranial pressure, then the operation of choice is the subtemporal decompression and drainage. If there are no definite localizing signs of the intracranial lesion, then the decompression should always be performed on the right side in right-handed patients, in order to lessen thereby any possible operative damage to the motor speech area of the left cerebral cortex. In patients having depressed fractures of the vault, showing definite signs of a high intracranial pressure, it is better surgical judgment to precede the elevation of the depressed area of bone by an ipsolateral subtemporal decompression, so that, when the depressed bone is removed, there will be little or no danger of the underlying cerebral cortex being damaged by its protrusion upward through the bony opening. As the subtemporal decompression exposes a comparatively silent area of the brain (a portion of the temporo-sphenoidal lobe), its protrusion and possible damage would not appear clinically; whereas a partial paralysis, impairment of sensation or of vision, might occur, and frequently does result from operations performed over the more highly developed areas of the cerebral cortex. Besides, the subtemporal route provides not only an excellent exposure of the middle meningeal artery and that portion of the brain so frequently involved in cranial injuries, but it affords drainage to the middle fossa of the skull (the chief intracranial cistern) at its lowest point in the base of the skull. Again, the thinness of the squamous portion of the temporal bone makes the operation a less difficult one technically. The vertical incision (and not the obsolete curved incision) should be used not only to render the operative hemostasis more effective, in that the trunk of the temporal artery is clamped at its lowest point in the very beginning of the operation, and thus there is no bleeding from its branches, but the vertical incision also permits the extensive removal of the underlying squamous bone not only down to a level with the base of the skull and thereby facilitating drainage, but also as widely as is p beneath the temporal muscle and yet the attachment of the temporal to the parietal crest is left intact, so that a firm closure of its separate cle-fibres is assured; this is a most important factor in patients havir intracranial pressure, as in brain tumor, where a cerebral hernia or might result from an imperfect closure of the temporal muscle and its The insertion of silver and celluloid plates and other foreign bodies b the scalp for the closure of cranial defects is to be most strongly cond

If the intracranial pressure is so high that the cerebral cortex t protrude through the bony opening, it is frequently wiser in s patients to perform a similar operation upon the opposite side of th immediately after the first operation. I have been obliged to do this five per cent. of the patients; they are the ones having swollen ede brains—"water-logged," as it were—where the drainage of blood an brospinal fluid is slight, and not sufficient to cause a marked decrease intracranial pressure. In some doubtful cases it is better judgment for one or two days, and even longer, before the second operation sidered advisable. The rubber tissue drains are usually removed on t or second day post-operative, and the hospital convalescence ordina quires at least ten days or two weeks. Naturally, these patients sho enter into their former active life for a period of three months, an longer; a too early return to the strain and stress of modern life pred them to many complaints, both subjective and objective. Repeated ex tions of the fundi of the eyes and of the superficial and deep reflexes a most important in estimating the physical normality of the patient.

Chronic Brain Injuries in Adults.—The end-results of patients brain injuries, with and without a fracture of the skull, have been an esting study. It has become quite a common belief that once a man ha "fracture of the skull," and then recovers, he is never the same person In 1912, I examined the records of three of the large hospitals of Ne City during the decade of 1900–1910; the mortality of "fractures skull" was 46 to 68 per cent.; the mortality of the patients operate was 87 per cent. This high operative percentage was due undoubtedly operation being postponed until the extreme stages of medullary co sion and edema, and also to the fact that the operation performed v "turning down" of a bone flap—a much more formidable procedur a subtemporal decompression—and then the bone itself replaced, even the benefits of a real decompression were thus prevented; besi many patients, the dura was not opened, and, as the dura is inel adults, therefore no adequate relief of the pressure could possibly tained. Of the patients, however, who were finally discharged as " or " cured," I was only able to trace 34 per cent., but of these 34 p of the total patients found, 67 per cent. of them were still sufferin the effects of the injury. The chief complaints were persistent head change of personality of the depressed or of the excitable type, ar emotionally unstable; early fatigue, making any prolonged physical c tal effort impossible, and thus the inability to work; lapses of memory of dizziness and faintness, and even epileptiform seizures in a sm tage of them. In examining the hospital records of the patients having se post-traumatic conditions, it was most interesting to ascertain that ese were the patients—and there were but few exceptions—who regained consciousness gradually after several days, and remained in the hospital for a period of four weeks and longer, whose charts made frequent mention of severe headache and a low pulse-rate of 60, and in some cases below 60—that is, the usual clinical signs of an increased intracranial pressure; an ophthalmoscopic examination had rarely been made. Many of these chronic patients still showed the results of the increased intracranial pressure in their fundi and at lumbar puncture, and these were the ones upon whom a cranial decompression, even at the late date of several years, caused a marked improvement. The operative findings were always associated with a "wet," swollen, edematous brain. Many of the so-called post-tramautic neuroses are, in my opinion, frequently superimposed upon this definite organic basis as the result of the brain injury. The treatment, therefore, of brain injuries should not be limited merely to the recovery of the patient, as far as life is concerned, but it should also be directed toward obtaining a normal individual, approximating as closely as possible the condition of the patient before the injury.

Gun-shot injuries, as well as stab-wounds of the brain, are usually associated with a penetrating fracture of the skull, and therefore may be considered in the same class as cerebral injuries following fractures of the skull. The greater danger of infection is present, however, and especially is this true when the missile has passed through the nasopharynx. Unless these patients are treated early, they rarely recover without serious complications. Particularly is this so when the dura and brain have been perforated—there being both a wound of entrance and of exit. These patients should all be treated as brain injuries having an increased intracranial pressure of sufficient degree to warrant the operation of cranial decompression. I have yet to see a penetrating gun-shot injury of the brain which did not cause a marked increase of the intracranial pressure, due to the resulting cerebral hemorrhage and edema, unless the cranial wounds of entrance and exit have been of unusual size to permit excellent drainage or the shock has been extreme. So that, not only is the operation of cranial decompression and drainage usually advisable to lessen this pressure, but also as a means of lessening the danger and even preventing a meningo-encephalitis, so frequent in the patients who survive the initial period of shock and active hemorrhage. Naturally, if the missile has passed through the basal ganglia, ventricles, the subtentorial tissues, and large intracerebral vessels, then the shock, with or without a large hemorrhage, is so rapid that these patients rarely survive a period of time sufficient to warrant any operative procedure; besides, if in severe shock, naturally no operation should be attempted, just as in brain injuries following head trauma. If the patient with a pulse-rate over 120 cannot react sufficiently to overcome this condition of shock, surely no operation will assist him. If the patient does survive the shock, then a decompression should be performed, and, if necessary, a bilateral decompression, and both the skull openings of entrance and exit should be enlarged with rongeurs, " cleaned " as well as possible, and rubber tissue drains inserted. By no means should the brain be probed c “ explored ” for bone and bullet fragments, as more damage, such as an i crease of the cerebral hemorrhage and especially the edema, as well as direct destruction of the delicate nerve-tissues, usually results from suc procedures. There is little danger from subcortical foreign bodies other tha that of infection, and the mere removal of the foreign body does not lesse that danger, as it would have occurred at the time of the injury. Such mec dlesome procedures, especially when the patient is in the stage of initi shock, merely hasten the death of the patient, just as in brain injurie following cranial trauma, if the patient is in severe shock, treat him fc shock, and “let him alone”—not even careful neurological examination to ascertain the exact cerebral status; such examinations of a patient i severe shock surely do not benefit the patient, and undoubtedly they lesse his chances of surviving the shock. If, however, the patient can overcom this condition of shock, then he should be most carefully examined, and t proper treatment of the local injury instituted as soon as possible.

On account of the serious and most extensive cranial injuries occurrin in the world war and resulting in large cranial and cerebral defects, ther has been a tendency to forget some of the underlying principles of brai surgery, as though the principles of neurologic surgery were not the same a in civil life. The methods may vary in individual patients, owing to the e treme character of many cerebral injuries in the war service, yet the ope ative treatment of these patients is based upon the presence of intracrani pressure, with the much greater danger of infection and loss of cerebr tissue; otherwise the treatment is essentially the same. To excise brai tissues freely, as if they were so much muscle or fat, when macerated an apparently infected, is undoubtedly necessary and advisable in selecte patients, but to state that cranial injuries in the war service should be treate in this manner, as though it were the usual method of treatment of brai injuries, and that this method should be used in civil life, cannot be co demned too strongly. Naturally, the greater danger of infection of wa wounds of the brain make these cases very serious ones indeed, and yet i the cardinal principle of lowering the increased intracranial pressure in th treatment of these selected patients by a large cranial decompression is ob served, even these patients will be given a greater and a definite chanc of recovery.

Brain Injuries in Newborn Babies and Children.—The symptoms an signs of brain injuries in newborn babies and children are in many ways s different from those following similar injuries in adults that it is necessar to devote to them a separate chapter.

Acute Brain Injuries in Babies.—In babies, owing to the open fo tanelles and to the greater elasticity of the dura, the immediate symptom and signs of brain injuries are often so mild that they are frequently ove looked, unless most careful and thorough neurological examinations ar made, and certain special aids of diagnosis utilized, such as the ophthalm scope and the examination of the pressure and of the cerebrospinal flui itself at lumbar puncture by means of the spinal mercurial manomete These intracranial lesions may escape serious attention for a period o nths in new-born babies and of even years in many children until the con- tion of cerebral spastic paralysis and its allied impairments, both physical nd mental, appear. Repeated lumbar punctures alone may be sufficient for drainage of the acute intracranial condition of hemorrhage and cerebral edema.

Chronic Brain Injuries in Children.—The remote effects of brain injuries in babies and children, such as spasticity, mental impairment in many patients, and frequently epilepsy, are merely reminders of the former intracranial damage, usually a supracortical hemorrhage and we should be most careful in our examinations and treatment to anticipate these frightful sequelæ. The older the children following intracranial birth traumata and the longer the period of time since the injury in the older children, the less hopeful is the prognosis. These late patients are derelicts, as it were, and can merely be improved; whereas if the condition of cerebral hemorrhage and edema is recognized as early as possible after the intracranial lesion has occurred, and if there is a marked increase of the intracranial pressure and the proper operative treatment of cranial decompression and drainage instituted, then in these patients so treated not only will the ultimate improvement be greater, and even a cure may be obtained, but also the immediate recovery of life be greatly enhanced. These lesions in babies and children have been so overlooked, and even neglected, that it seems advisable to report a large number of these cases in detail. Naturally, the older the child, the more do the symptoms and signs of an intracranial lesion resemble those occurring in an adult, and yet the brain in children under the age of puberty is so adaptable to changed conditions, and to a certain extent less delicate, that even a high degree of intracranial pressure, due to hemorrhage and cerebral edema, may present clinically few signs of its presence, and in many patients it can be withstood, and undoubtedly is successfully drained, by natural absorption alone. This fact should always be remembered in the treatment of brain injuries in children, so that no operation should be advised unless the intracranial pressure in these patients is very high, and when it is doubtful whether the child can "take care of" this increased pressure alone and even with the aid of repeated lumbar punctures daily. Thus does the treatment of brain injuries in children differ from that in adults. Naturally, just as in adults, all traumatic depressions of the vault, with or without a definite fracture of the bone itself, should be elevated or removed; in babies, the use of forceps in difficult labor frequently produces a definite depression of the vault without a fracture of the bone itself, owing to its greater resiliency, and unless this depressed area of bone is elevated or removed, the danger of future cerebral impairment is great indeed. It is frequently not necessary to open the dura in these cases of local depression of the vault in babies, as subdural and supracortical hemorrhage rarely results from it. Naturally, in cases of doubtful subdural hemorrhage and cerebral edema, the dura should always be opened through a subtemporal decompression, just as in adults having an increased intracranial pressure associated with a depressed fracture of the vault, and then the local bony depression elevated or removed.

CHAPTER II

GENERAL CONSIDERATIONS

THE entire subject of brain injuries has been so obscured and confused by the question, "Is a fracture of the skull present?", as though the presenc or not of a fracture of the skull were the important factor of cranial injuries that the progress in the diagnosis and treatment of brain injuries has no been commensurate with the advance made in the other branches of medicin and surgery. Before the extensive use of the Röntgen ray, patients having brain injuries, if the depressed fractures of the vault are excluded, wer possibly more *rationally* diagnosed and treated in many hospitals than they have been within recent years under the most modern developmen and accuracy of röntgenograms; so frequently, it is still asserted, following cranial injuries with negative röntgenograms, that "as no fracture of the skull is revealed by the X-ray, the condition is not a serious one," and the converse with positive röntgenograms that, "the condition is a most seriou one in the presence of such an extensive fracture of the skull." These statements are also frequently made in court, while the positive X-ray pictures are shown impressively to the judge and jury.

It is now well known that severe brain injuries need not be associated with a fracture of the skull; that a positive röntgenogram (unless it discloses a depressed fracture of the vault) in no way lessens the effectivenes of the expectant palliative treatment, but that it frequently aids this medical treatment by permitting the drainage of intracranial hemorrhage and cerebrospinal fluid through the lines of fracture into the subcutaneous tissue of the scalp, where hematomata of varying sizes may be formed, or into the nasal cavity and most frequently into either auditory canal. In thi manner, the increased intracranial pressure is lessened so that the mechanical operative relief of the intracranial pressure, whether due to hemorrhage or cerebral edema, is rendered unnecessary and thus a cranial operation is avoided; and on the contrary, if there is no line of fracture of the skul present, then whatever intracranial hemorrhage or cerebral œdema occurs following a cranial injury is in no way drained and lessened other than by the natural means of absorption through the cortical veins, sinuses and possibly lymphatics; and should this method alone of lessening the intracranial pressure prove insufficient, then the operative mechanical method of drainage would be advisable in order to obtain the best result—not only the recovery of life of the patient, but, of almost equal importance in many of the patients, the return to a normal condition, both mentally and physically, as before the cranial injury. To advise a cranial operation just because the röntgenogram reveals a fracture of the skull (in the absence of a depressed fracture of the vault) is most strongly to be condemned—no matter how extensive the line or lines of fracture are; in fact, the more extensive they are the greater the probability of their permitting the intracranial hemorrhage and cerebral edema to escape extracranially and thus the serious condition of intracranial pressure and medullary compression be avoided. Lines of cture, however, extending into the posterior fossa subtentorially are ually most serious types of fracture on account of the greater danger of subtentorial hemorrhage and edema producing a direct compression upon the medulla itself, although even in these patients this increased subtentorial pressure is occasionally entirely relieved by the drainage of the hemorrhage and cerebrospinal fluid through the lines of fracture and into the tissues at the base of the neck. Conversely, not to advise a cranial operation in the presence of a high intracranial pressure, whether due to hemorrhage or cerebral edema, merely because of the absence of the usual and historical signs of a fracture of the skull, such as a bleeding and discharge of cerebrospinal fluid from the nose and ears, subconjunctival and mastoid ecchymoses, and a line of fracture of the skull is not revealed in the röntgenograms, is equally to be condemned. Most of the mistakes in the diagnosis and treatment of brain injuries occur in this class of cranial injuries, and especially in those patients of middle-age and older, associated with other conditions, particularly chronic alcoholism, chronic nephritis, and arteriosclerotic conditions; it is in these patients that the acute condition of "wet" brain (acute cerebral edema) occurs so easily and most frequently following cranial injuries of apparently trivial character and not even of sufficient force to produce a fracture of the skull. If careful and repeated examinations are made, however, the preliminary stages of these conditions can be recognized early, the proper treatment instituted and thus the advanced and extreme condition of "wet" brain can be early relieved, if not even anticipated and avoided.

Careful röntgenograms, however, should be made in different planes of all patients having cranial injuries, no matter how trivial apparently, in the knowledge that if a line of fracture is disclosed, then the patient will receive much more careful examinations and treatment—the doctor realizing that the cranial injury was of sufficient force to fracture the skull, with possibly intracranial damage and complications, so that the after-treatment and advice will also be more careful; naturally depressed fractures of the vault must be ascertained in all cases of cranial injury and the appropriate treatment of elevation or removal of the depressed area of bone early performed. The treatment of patients having cranial injuries, therefore, should not be delayed in the absence of röntgenograms, and especially when associated with a high intracranial pressure, and the presence or not of a fracture of the skull (if the depressed fractures of the vault are excluded) should not in any way predispose the doctor toward the method of treatment—whether the expectant palliative treatment or the operative method of subtemporal decompression and drainage is adopted. No cranial operation should ever be performed upon these patients in the absence of high intracranial pressure unless there is a depressed fracture of the vault; merely a slight increase of the intracranial pressure, whether due to hemorrhage or cerebral edema, is usually "taken care of" by the natural means of absorption alone, and if there is no increase of the intracranial pressure at all, naturally no operation of cranial decompression is necessary. The operation is to relieve intracranial pressure both by a simple decompression and by drainage; but if there is no pressure present, it of cours relieve it, and therefore the operation would be an unnecessary and meddlesome procedure—at best the operation should not then be called a "decompression." This criticism is applicable to the so-called "decompression and drainage" of cases of internal capsular hemorrhage occurring in the usual type of apoplexy—and even in the absence of an increased intracranial pressure!

The presence of paralyses of the cranial nerves, in the absence of a marked increase of the intracranial pressure, is due to a direct injury to the cranial nerves themselves, and in conditions of paralyses of the extremities the usual cause is a cerebral laceration and contusion. Of the cranial motor nerves, the motor oculi (III), the abducens (VI), and particularly the facial (VII), are most frequently involved, the impairment being either a severance of the nerve itself and therefore a permanent paralysis, or more frequently merely a compression of it by hemorrhage and, in the case of the facial nerve, in its bony canal of the aqueduct of Fallopius, by edema and therefore usually a temporary impairment only; whereas in paralyses of the extremities in the absence of a definite increase of the intracranial pressure, a cerebral laceration of the cortical and subcortical type should be suspected. In conditions of hemiplegia (if no increased intracranial pressure is present), a lesion of the internal capsule itself must be considered. Naturally, in these acute paralytic conditions in the absence of a high intracranial pressure, no cranial operation is indicated. (It is possible for a small cortical hemorrhage to be the cause of the monoplegia and yet no marked signs of an increased intracranial pressure be present. In carefully selected cases a cranial operation might be advisable for these patients, although the majority of them recover, without any cranial operation, by the means of natural absorption alone.) It is a rule, therefore, only in these patients having paralysis in whom there is a marked increase of the intracranial pressure, that a cranial operation is indicated.

The above method of procedure is equally true of patients having convulsive seizures; if there are no marked signs of an increased intracranial pressure, then it is unwise to advise a cranial operation, unless the convulsions are of a very severe and persistent character. One or two attacks within several days following an acute cranial injury should not make a cranial operation advisable, especially in the absence of an increased intracranial pressure. (Naturally, if a depressed fracture of the vault should be ascertained, it should be immediately elevated or removed.)

TYPES OF FRACTURES OF THE SKULL

Fractures of the skull may be classified briefly according to their location; "direct" fractures occurring at the immediate area of contact, and the "indirect" fractures occurring at various distances from this area of contact.

I. **Direct Fractures.**—The "direct" or local fractures may consist of a break of the outer table or of the inner table of the skull, or of both if the impending force is sufficiently strong. If the surrounding outer table is broken, a partially depressed fracture may result; if the surrounding inner table is also broken, then a completely depressed fracture is possible; if more than one fragment is present, the term "comminuted" fracture may used. (Figure 1 demonstrates the various degrees of "direct" fractures.) irty-six of my operated patients represented different degrees of direct r local fractures.

II. **Indirect Fractures.**—The "indirect" fracture is usually a linear one—extending away from the area of contact and most frequently down into the base of the skull, especially into the middle fossa. My experience has been that it is rare for a fracture to be limited to the vault alone, whether the fracture be a simple linear one—a "crack"—or one with depression; it will usually be found that a line of fracture extends from the thicker bone of the vault down to the thinner bone of the base, and this is what we should naturally expect. Eighty-five of my operated patients were of this type.

Many theories have been evolved to explain this tendency of fractures to radiate to the base. It was a common observation that if the cranial vault was struck by a fairly pointed object, then a localized depression or even a perforation would result, and possibly a line of fracture would extend downward into the base; however, if the cranial vault was struck forcibly by a blunter, wider surface, then there might not be a localized depression, but a line or lines of fracture would extend downward into the base and up to the opposite side, the lines of fracture tending to merge at a point directly opposite the area of contact by the object. This radiation of the lines of fracture and their tendency to merge on the opposite side, producing what has been termed the fracture of "*contre-coup,*" are most satisfactorily explained, in my opinion, by the "bursting" effect of injuries of the cranial cavity. When the head is forcibly struck against a hard object, the point of contact tends to be approximated to its pole on the opposite side, and so mechanically there is a tendency for a line of fracture to occur in one of the meridians extending from the point of contact to its opposite pole; as the base, especially its middle fossa, is much thinner and weaker than the bone comprising the vault of the skull, naturally the line of fracture extends downward into the base, and the so-called "fracture of the base" results. The line of fracture may, however, merge into one of the sutures of the vault of the skull, producing the so-called "fracture by diastasis," which most frequently occurs in children.

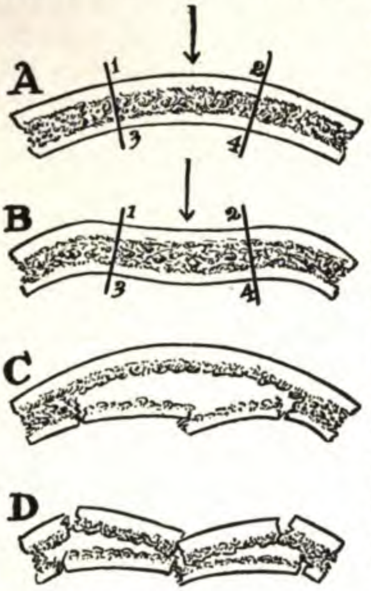


FIG. 1.—Local depressed fractures of the vault; the mechanics of their production. A, the normal vault, with its outer and inner table, and the interposed diploë The arrow indicates the force applied at the point of contact, and the lines 1–3 and 2–4 are placed at right angles to both the outer and inner tables; naturally they converge. B, the force applied at the point of contact produces a depression of both the outer and inner tables, and especially of the inner table, as shown by the divergence of the lines 1–3 and 2–4. C, the inner table may fracture and a fragment may be depressed, and yet the outer table may remain intact, either depressed or, as illustrated here, in its original position. D, a complete depressed fracture occurs when the force applied is sufficient to cause a fracture not only at the point of contact but also at the margin of the depressed area; the line of fracture of the inner table is always beyond that of the outer table.

These lines of "bursting" fractures of the skull usually extend downward into the middle fossa through the petrous portion of the temporal bone, rupturing the tympanic membrane and thus allowing blood from the middle ear, and even cerebrospinal fluid, to escape at the external auditory meatus. If the line of fracture extends into the anterior fossa, then the cribriform plate of the ethmoid bone, being the thinnest and weakest portion, is usually "cracked," producing bleeding from the nose. Mere bleeding from the nose or from the ears, however, does not necessarily indicate a fracture of the base of the skull, because the blood may result from a local injury to the nose itself or to the external auditory canal; if, however, cerebrospinal fluid is also observed, then a fracture must be present. Extreme care should be used in ascertaining a rupture of the tympanic membrane because of the great danger of infection; it is much wiser merely to wipe the external auditory meatus with sterile cotton and not attempt to introduce instruments for an otoscopic examination; naturally, if there is no bleeding from the ear, an otoscope can be used without danger of infection, and frequently blood in the middle ear will be diagnosed by the bluish color of the tympanic membrane, which has remained intact.

In this series of 155 operated patients, the area of contact was in the parieto-temporal region in 71, and in 49 of these a line or lines of fracture extended downward into the middle fossa or forward into the angular process of the orbit of the same side. In 68 of the patients having a fracture of the base of the skull and in whom no signs of fracture of the vault were ascertained, in performing a subtemporal decompression, I found a line of fracture extending upward into the vault in 25 of them. Fractures of the frontal region most frequently radiate into the orbital bones and into the anterior fossa, producing marked orbital signs and a hemorrhage and leakage of cerebrospinal fluid into the nasal cavity.

Fractures of the posterior portion of the vault beneath the tentorium usually send lines of fracture, when they do radiate, downward toward the margin of the foramen magnum, and frequently beyond it into the basilar process. This type of fracture is the most serious of all cranial fractures, due chiefly to the great danger of medullary compression and collapse resulting from the pressure of hemorrhage or edema beneath the tentorium directly upon the medulla. The signs of medullary edema in these patients usually appear very soon after the injury, and death frequently occurs within three hours. A subtentorial decompression in these patients offers what little chance there is for recovery. Eight of the operated patients that died revealed at autopsy this type of fracture of the occipital bone and its basilar process and associated with large subtentorial hemorrhage.

It is interesting to note that a most common site for "direct" depressed fractures is in the upper posterior portion of the vault, about the posterior fontanelle, and yet the lower occipital area surrounding the foramen magnum is so well protected by large bony buttresses that the lines of fracture in these patients radiate most frequently forward into areas of comparative safety rather than backward into the occipital bone beneath the tentorium—a most dangerous area.

CHAPTER III

THE SYMPTOMS AND SIGNS OF ACUTE BRAIN INJURIES

I. **Symptoms of Acute Brain Injuries.**—The initial symptoms of acute brain injuries are few. Most of these patients are unconscious for varying periods of time, so that their subjective sensations, if any, are not communicated. Upon being aroused from the condition of unconsciousness, which usually occurs within several hours after the cranial injury, or shortly after the decompression in the operated patients, these patients are in such a stuporous, drowsy condition, and their mentality is so confused, that they have great difficulty in making known their complaints. If they do recover consciousness sufficiently to make themselves understood, then the chief outstanding complaint, which is always present, is headache of varying character and degree. It frequently happens that many patients having most severe brain injuries are not even rendered unconscious, or, if so, then for several minutes only; these patients, however, all complain of the most intense piercing headache, which usually increases as their entire consciousness more fully returns.

1. *Headache.*—Headache of a "throbbing," "beating" character is practically always present. At times it is only a dull, heavy feeling in the very mild cases, while in the patients who still remain conscious it is very severe and intense—the typical "splitting" headache; the resulting restlessness may be extreme. The headache of these patients is now known to be due to tension upon the dura resulting from the mild increase of the intradural pressure from hemorrhage, and, more usually from a cerebral edema of varying degree—that is, an increased amount of cerebrospinal fluid, due to a temporary cessation of its excretion into the cortical veins, sinuses, etc. In the mild cases, the headache persists for several days only, and gradually lessens as the increased amount of cerebrospinal fluid, clear or blood-tinged, is absorbed by the cortical veins and sinuses. If, however, the cranial operation of subtemporal decompression is necessary to relieve the high intracranial pressure, then the headache disappears, almost immediately following the operation. This observation is most impressive and striking, and it is the usual history.

2. *Nausea and Vomiting.*—Nausea is very common, and if the injury has been received two or three hours after a meal, then vomiting may occur. Too much importance, however, has been placed in the past upon the symptom of nausea; in my experience, it is associated with any condition producing shock, whether the injury be cranial, abdominal, or elsewhere. It is no index of increased intracranial pressure unless in the extreme cases, and in subtentorial lesions about the medulla, when it is always associated with vomiting. Chronic alcoholism predisposes many of these patients to the excessive nausea and vomiting which otherwise would only occur infrequently in the more temperate patients having the cranial injuries.

II. **Signs of Acute Brain Injuries.**—It is upon the signs of brain injuries that the greatest importance must be placed in their diagnosis and treatment. The signs of cranial injury are many and multiform, whereas those of brain injury are comparatively few, but most important. It must always be remembered that a brain injury is not necessarily present merely because the vault or the base of the skull is fractured or the head is badly lacerated (Fig. 2), and conversely, that a brain injury may, and frequently does, occur without there being a fracture of the skull or the other signs of an external cranial injury (Fig. 3).

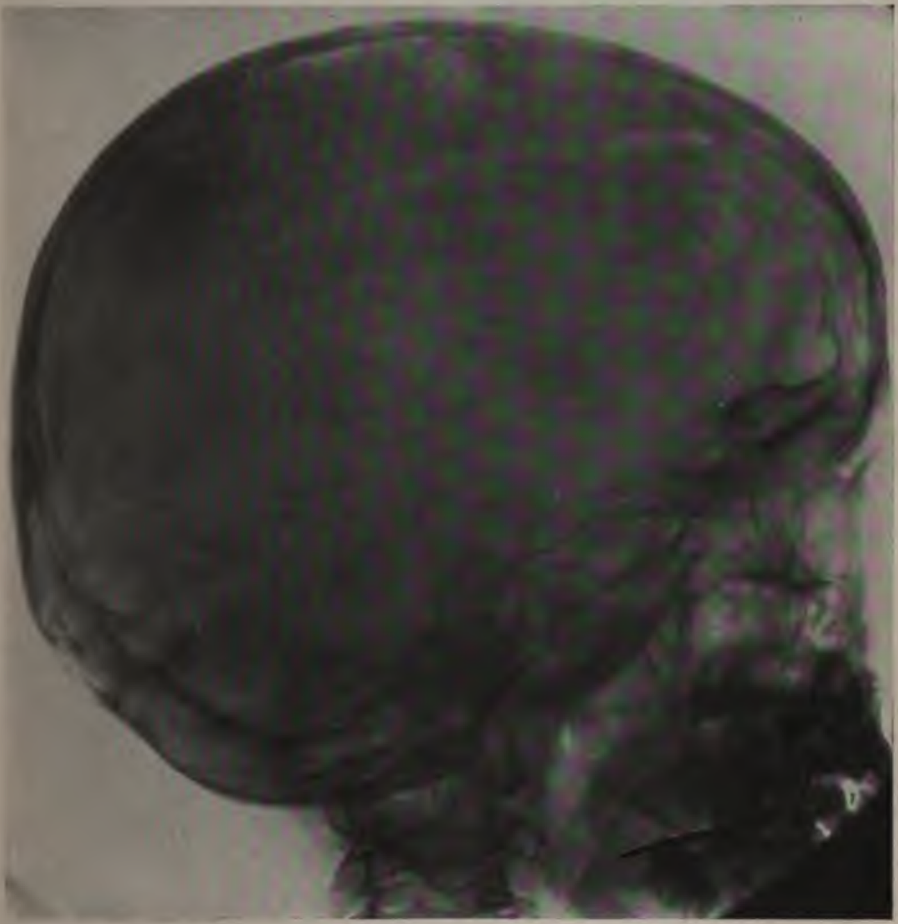


FIG. 2.—William W. Large linear fracture of the vault—at least one-quarter of an inch in width and extending from the right frontal bone backward into the right occipital area, in a boy of eighteen years of age, who walked into the accident room of the hospital complaining of a "soreness in the head" following a fall of almost one hour previously. Upon examination, there were no positive neurological signs; the ophthalmoscopic examination was negative, and the spinal mercurial manometer registered a normal pressure (8 mm.) of the cerebrospinal fluid, which was clear. No operation; expectant palliative treatment. Uneventful recovery after a period of ten days in the hospital. Present condition—excellent.

A. LOCAL SIGNS.—A cranial injury of sufficient force to produce a brain injury, with and without a fracture of the skull, usually damages the tissues of the outside of the head to a greater or less extent. Such confusing contusions, however, must be rigidly excluded in making a diagnosis

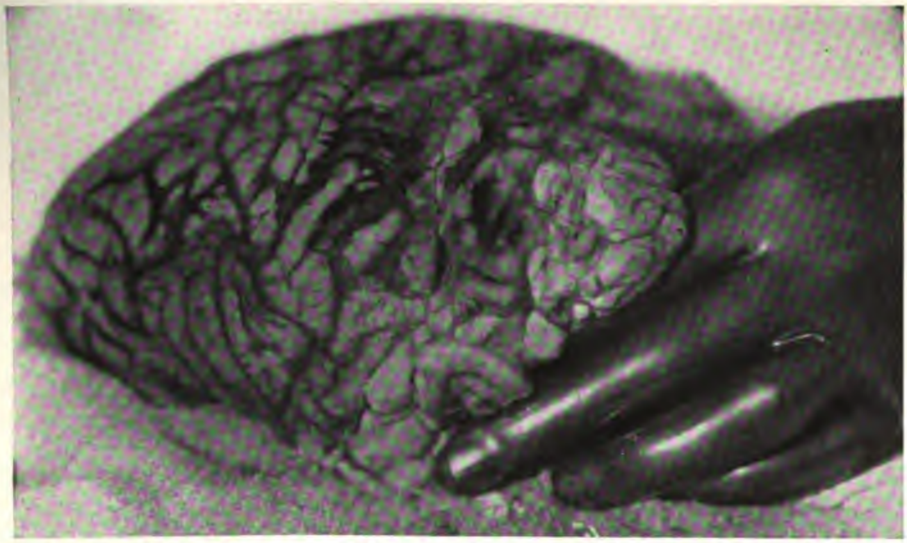


FIG. 3.—Henry W. Extensive supracortical and subarachnoid hemorrhages associated with acute medullary edema, but *no* fracture of the skull, in a man who had fallen, while asleep, from a fire-escape to the ground—a distance of twelve feet; marked stupor upon admission to the hospital two hours after the injury (2 A.M.). As there were no signs of a "fracture of the skull," such as bleeding from the nose, mouth or ears, et cetera, and since the general condition of the patient "seemed good," it was decided to "wait until morning" and then have an "X-ray picture taken." At 7 A.M. (5 hours after the injury and 3 hours after admission), the patient suddenly developed extreme signs of an acute medullary edema—the pulse- and respiration-rates quickly ascending to 150 and 50 plus, respectively, so that death occurred at 10 A.M. (8 hours after the injury and 6 hours after admission to the hospital). An autopsy did not reveal a fracture of the skull. Careful repeated examinations could have, at least, anticipated this unfortunate result.

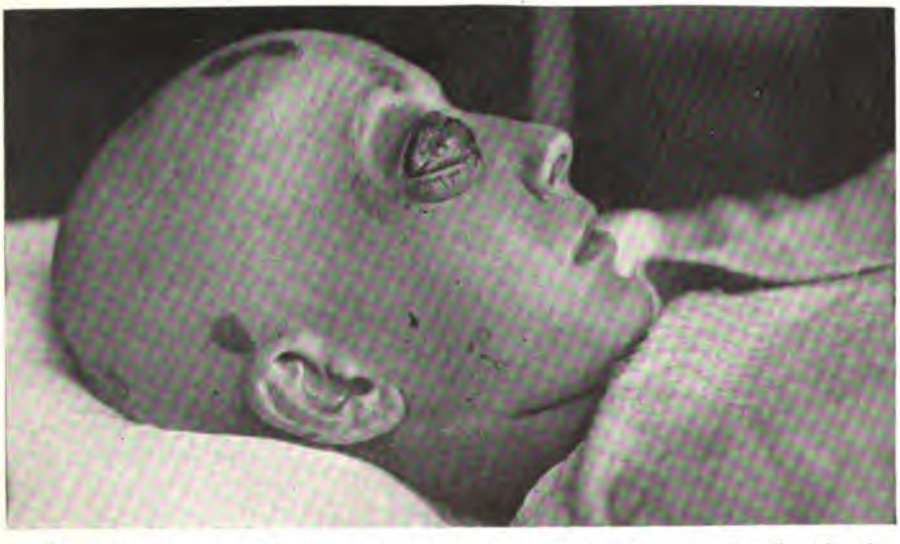


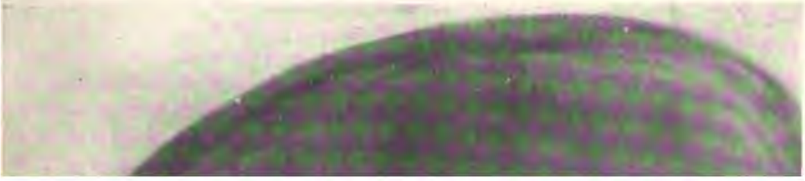
FIG. 4.—Theodore A. Multiple hematomata of the scalp and a large hemorrhage into the right orbit, with resulting exophthalmos in a youth sixteen years of age, with profuse bleeding from the left ear and left nostril upon admission to the hospital, and yet no fracture of the skull could be demonstrated either by X-ray or at the later operation of right subtemporal decompression; a very "wet" edematous brain producing signs of a high intracranial pressure was revealed and drained—permitting an excellent recovery. Otoscopic examination disclosed an intact left tympanic membrane and merely a laceration of the posterior wall of the left external auditory canal.

of a fracture of the skull. Ecchymoses about the orbits and mastoid regions, and hemorrhage from the nose, mouth and ears, are frequently the result of local injuries, and in no way associated with a fracture of the skull (Fig. 4). Hematomata of the scalp, especially if subpericranial, may easily simulate depressed fractures of the vault; and the reverse is also true in that linear fractures of the vault of varying degree are frequently concealed beneath hematomata (Fig. 5). In doubtful cases of depression of the vault, the head should always be shaved, and an exploratory incision through the scalp made to ascertain the presence or not of a bony depression; a careful bimanual palpation of the head is very important in all of these patients.

Any bleeding coming from the nose, throat or ears should have its source investigated, but always under the most rigid asepsis. If cerebrospinal fluid is observed, then a fracture of the skull must be present. Subconjunctival hemorrhages, as well as ecchymoses in the mastoid areas, appearing soon after the accident, without a local contusion being present, are suggestive of a basal fracture. If these signs appear after one or two days, they are especially indicative. It is possible, however, to have extensive subconjunctival hemorrhages, and yet there is present no fracture of the orbital bones. This observation has been frequently confirmed at autopsy.

In many fractures of the skull extending into the orbital, temporal and occipital bones, the escape of cerebrospinal fluid may be so free that the tissues about the orbits, ears and in the occipital region will become markedly edematous and boggy. A mere ecchymosis of the orbit is, however, of no diagnostic value, since any injury to the anterior portion of the scalp will produce the typical "black eye."

An ecchymosis of the scalp itself does not indicate the presence of an underlying fracture of the vault; but if the oozing of blood is subpericranial, then an underlying or adjacent fracture is very probable. Particularly is this true if the ecchymosis is in the temporal muscle beneath the temporal fascia, and especially if the overlying scalp is normal in appearance. In these patients the presence of an ecchymosis or free blood in the temporal muscle beneath the temporal fascia indicates the presence of a fracture of the underlying squamous bone most frequently, or of any part of the vault included within the limits of the attachment of the temporal muscle. This observation has been repeatedly confirmed, in 61 patients in all, both at the operation of cranial decompression in the subtemporal region and also in the other cases at autopsy. Careful bimanual examination of this temporal area will often reveal a sort of crepitus and indefinite "crackling," due to the digital compression of the free blood enclosed in the temporal muscle by the intact temporal fascia. A röntgenogram is of great assistance in confirming this diagnosis, which is of importance owing to the frequent complication in these patients of the adjacent middle meningeal artery and the formation of a resulting extradural clot of varying size and compressive effect upon the underlying cerebral cortex. These fractures of the squamous bone, fortunately, extend very often into the middle ear, and, as the tympanic membrane is usually ruptured, the intracranial hemorrhage can escape through the external auditory canal,



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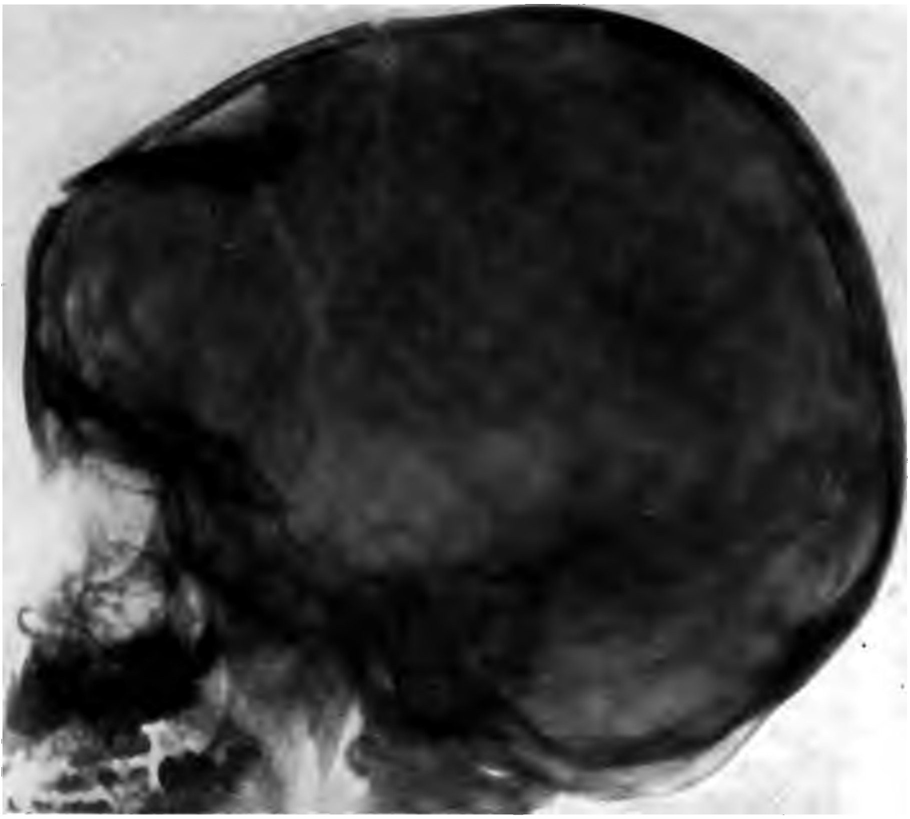

Fig. 6.—Harriet G. This patient of 43 years of age slipped and fell headlong from the front door-steps to the pavement—a distance of four feet; no loss of consciousness—"merely dazed," and that afternoon, she came to the out-patient department of the hospital by trolley-car on account of the "bump" over her forehead, which was gradually enlarging, and "a dull aching in the head." The hematoma was so tense and extensive that palpation and the routine bimanual examination, in the presence of her hair, were negative. She was permitted to return to her home and advised to "come for an X-ray picture in the morning." The huge depressed fracture of the vault, and the radiating linear fracture to the base, were most instructive, and fortunately in this patient an excellent result was obtained by performing a right subtemporal decompression, first to lower the general increased intracranial pressure of blood-tinged cerebrospinal fluid, and then the local depressed area of bone was removed; the underlying dura had been lacerated and even some cerebral tissue was lying at the site of the depression. The present condition of the patient, thirty-four months after the injury, is normal. The value, however, of an early röntgenogram in this type of patient is obvious.

Röntgen Rays.—Röntgenograms in cranial injuries are of great value in the accurate *diagnosis* of fractures of the skull, but it is only in occasional patients having brain injuries that X-ray pictures are of any great aid in the *treatment* of these patients. Naturally, questionable depressed fracıres of the vault should always be pictured, and I believe that all cranial ınjuries of any severity, and in many cases of even mild degree, should have a röntgenogram taken in two or more planes for fear a trivial "bump" may be of greater severity than the history might indicate, and thus the appropriate careful treatment be neglected, or at least delayed (Fig. 6). But to insist in the more serious traumatic conditions of acute intracranial lesions that an X-ray picture be taken, even if such a procedure should delay and even postpone the operative treatment of the condition, such as "waiting overnight" in order to have a picture taken, is, in my opinion, not only poor judgment but an absurd and dangerous attitude. The sooner it is generally realized that the treatment in brain injuries (excluding depressed fractures of the vault) is not directed so much toward the ascertaining of the presence or not of a skull fracture, and its location and extent, as toward the lessening of the effects of the cranial trauma upon the intracranial contents—whether there is a fracture or not—just so much sooner will these patients receive a rational treatment. The mere fact that there is a fracture or "crack" in the skull, as shown by the röntgenogram, is no reason to advise an operative procedure, and, conversely, the absence of any definite fracture being revealed by the X-ray is no reason that an operation to lessen the intracranial pressure due to hemorrhage or edema may not be necessary.

Linear fractures of the vault occur much more frequently than is commonly believed and recognized. Routine röntgenograms are now taken of all my patients who have had a "bump" upon the head—at least two views: antero-posterior and lateral—and it is surprising the number of latent fractures of the vault which are thus disclosed. The effect may be of a trivial character, and yet when we know that a fracture of the vault is present, the examination and treatment of such a patient are undoubtedly more careful, and the prognosis is more guarded. Many of these patients have walked into the accident room of the various hospitals, the scalp not even being lacerated, and have complained merely of the "bump" and a slight headache; the latter might not continue longer than three or four days under the routine expectant palliative treatment. The usual sites for these latent fractures of the skull are the squamous portions of either temporal bone and the greater wings of the sphenoid bone posterior to the external angular process of the orbit (Fig. 7). Both sides of the head should be exposed. Stereoscopic views are very helpful.

The interpretation of röntgenograms of basal fractures is very baffling, and it is frequently impossible even to secure satisfactory pictures. The bony irregularities of the base of the skull, and the difficult position necessary to obtain the proper plane in order to facilitate the exposure of the fracture—these factors make the X-ray pictures of very doubtful value. A negative picture does not by any means indicate that a fracture does not exist. Fractures about the foramen magnum may be better photographed at times through the open mouth.

Naturally, the treatment of these patients remains the same, whether there is a fracture or not, a decompression being considered only in the presence of marked intracranial pressure. The operation is not to remedy

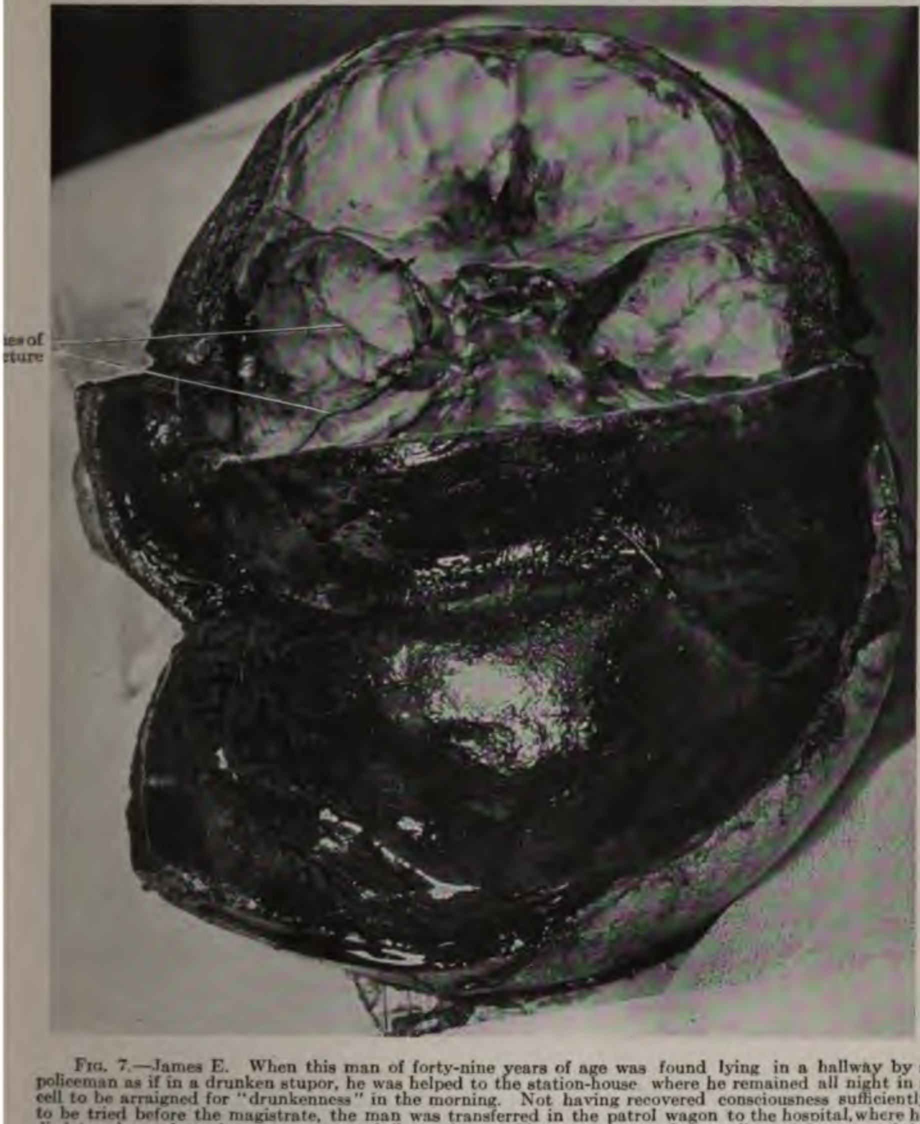

FIG. 7.—James E. When this man of forty-nine years of age was found lying in a hallway by a policeman as if in a drunken stupor, he was helped to the station-house where he remained all night in a cell to be arraigned for "drunkenness" in the morning. Not having recovered consciousness sufficiently to be tried before the magistrate, the man was transferred in the patrol wagon to the hospital, where he died two hours later from an acute medullary edema. The autopsy revealed two lines of fracture of the base of the skull—an irregular oblique one in the left greater wing of the sphenoid bone and another one along the crest of the left petrous bone but not into the middle ear; the left tympanic membrane was not ruptured—therefore, no bleeding or discharge of cerebrospinal fluid from the left ear. The brain itself was very "wet" and edematous with much free blood subtentorially about the medulla. Intracranial injuries are commonly obscured and concealed by alcoholism, and the most careful examinations are necessary to differentiate these conditions early—in order to be of value to the patient.

the fracture (unless it be a depressed fracture of the vault), but to offset the results of the fracture and the injury to the brain. In my opinion, all patients having possible brain injuries should be treated by the expectant palliative method, whether a fracture of the skull is present or not, and a decompression advised only when this method fails to prevent an increasing intracranial pressure, as shown by the careful and repeated neurological examinations, especially the use of the ophthalmoscope, and confirmed by a measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer.

Fractures of the occipital bone, subtentorially and radiating downward to the foramen magnum and even beyond, along the basilar process, are the most serious of all cranial fractures. The great danger of direct pressure upon the medulla itself, due to any resulting hemorrhage or edema subtentorially, is the complication most to be feared, and the one that usually terminates fatally within several hours unless it is of very mild degree (Fig. 8).

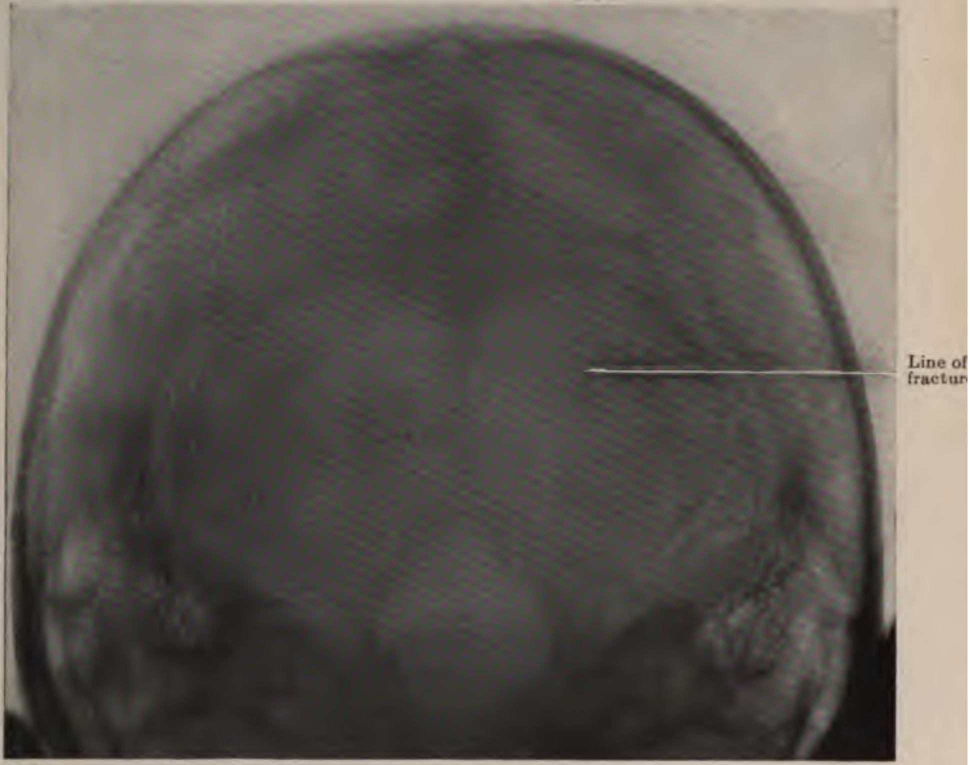

FIG. 8.—Esther R. This patient of twenty-four years of age fell while skating upon the ice, striking the back of her head. No immediate loss of consciousness; was able to walk home and to eat at the dinner table in spite of a "dull headache." Vomiting occurred two hours later, and at three o'clock (six hours after the injury), it was found that she could not be aroused to consciousness; her pulse was then fifty-four and respirations twelve and of a Cheyne-Stokes character. Upon admission to the hospital one hour later, an X-ray picture revealed a narrow linear fracture extending vertically downward and backward from the right parietal bone through the right half of the occipital bone into the right margin of the foramen magnum. The condition of the patient quickly changed after admission to the hospital in that the signs of medullary compression merged into those of an acute medullary edema—a rapidly ascending pulse- and respiration-rate, so that the patient was *in extremis* before the preparations for the operation of suboccipital decompression could be made. The autopsy revealed a large subtentorial hemorrhage directly compressing the medulla. An earlier operation would have afforded this patient a definite chance of recovery; the delay necessitated by an X-ray picture was inexcusable.

Fractures extending into the nose, mouth or ears, so that the escape through them of intracranial hemorrhage and cerebrospinal fluid is possi- frequently facilitate the expectant palliative treatment by permitting any increase of the intracranial pressure to be lowered by this means of drainage—a sort of "natural" decompression; the danger of infection extending intracranially through this line of fracture, and thus producing a purulent meningitis and meningo-encephalitis, is a slight one, unless meddlesome procedures are used, such as attempts to "swab" and "clean out" and theoretically sterilize the auditory canal and nares, and in this manner infective organisms are introduced through the line of fracture. The "snuffing" of aseptic solutions into the nose, and the irrigation of the ears of these patients, cannot be too strongly condemned. Moist aseptic gauze pads, loosely placed over the nares, so that the patient breathes chiefly through the mouth, and a similar gauze pad over the lobe of the ear, and not so tightly as to block the aural discharge, usually suffice to lessen the danger of a possible infection. In this series of patients having cranial fractures which extended into the nose, mouth or ears, a resulting infection occurred in only 4.6 per cent. of them (Fig. 9). The use of urotropin internally is no longer advocated, since it has been determined that urotropin is only effective in acid media, such as the urine, and not in neutral or alkaline media, such as the cerebrospinal fluid.

The most extensive linear fractures of the skull occur in children, and frequently with little or no apparent damage to the enclosed brain. So much drainage of the intracranial hemorrhage and increased cerebrospinal fluid into the nose, ears and tissues of the scalp and neck, is thus afforded in so many of these patients that they really "decompress" themselves, and so an excellent recovery is obtained without the necessity of a cranial operation.

B. GENERAL SIGNS.—1. *Shock.*—It is infrequent for cranial injuries to occur and not be associated with more or less shock, while it is most rare for brain injuries of any severity to exist without there being present the initial complication of shock. Only too frequently the condition of initial shock in these latter cases is so extreme that the patient is unable to survive it, and an early death results from the shock alone. These fatal cases of traumatic shock may occur from any severe bodily injury, and particularly of the abdomen and chest, and yet it is in brain injuries that the condition of initial shock is of the greatest importance and danger. If the patient, following a cranial injury, is in a severe state of shock, then all efforts should be directed toward the overcoming of the shock—no prolonged neurological examinations made, but the immediate use of measures to combat the shock, such as external warmth to the body (heated blankets, hot-water bottles, etc.), repeated rectal enemata of hot black coffee, absolute quiet, and codeine or morphine, if necessary, for restlessness. In this severe condition of initial shock, the chief concern of the physician is not whether a definite brain injury or a fracture of the skull is present or not, or whether there is a facial paralysis or a Babinski reflex present or not, or whether there is a depressed fracture of the skull or bleeding from the ear through a ruptured tympanic membrane, and many other data and observations of value *later* to both the patient and the doctor, but rather the early recognition of the severity of the shock and the appropriate treatment immediately instituted and then—the patient "let alone" until the condition of initial shock is survived, no matter how badly the skull is fractured, how pronounced the hemiplegia may be, or how "typical" the double Babinski may appear. If the patient cannot survive the shock, it will be of no value or interest to him whether the skull was fractured or not, or whether Babinski reflex was present or not.

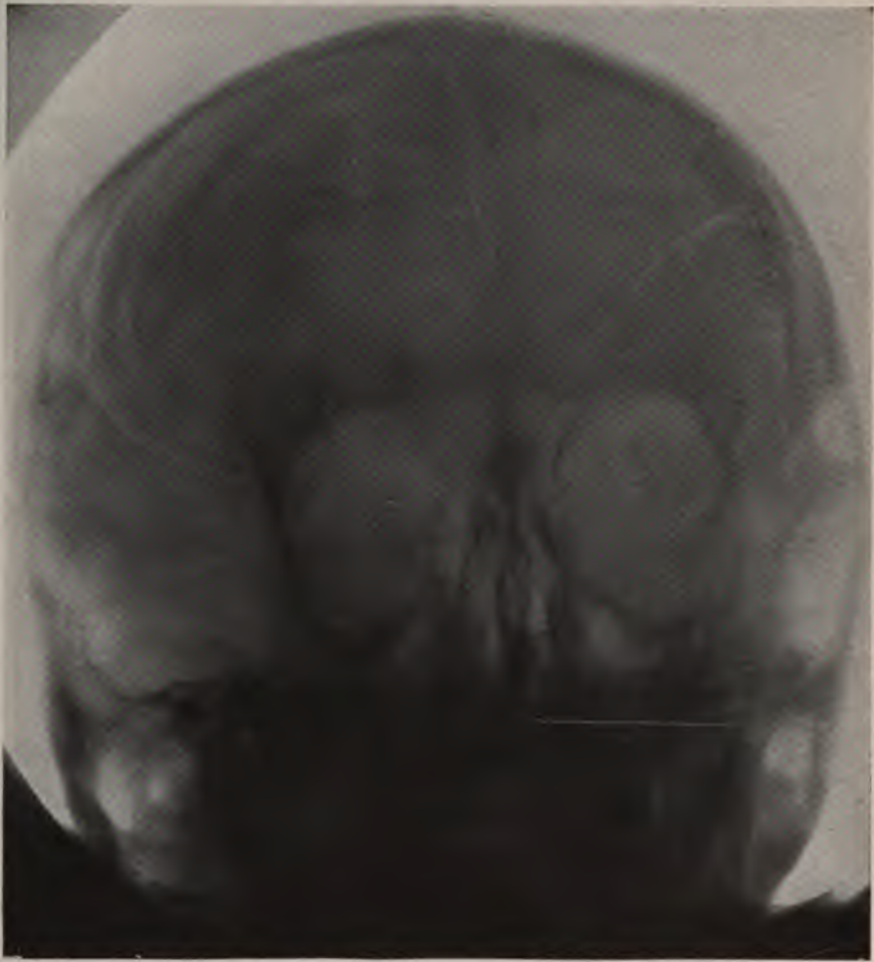

FIG. 9.—Patrick K. Multiple radiating linear fractures of the vault extending forward into both orbital plates and nares, in a youth of eighteen years of age, who had been struck by an automobile; only a momentary loss of consciousness. Profuse and continued nose-bleed for three hours; a "watery fluid" flowed out of the nose in spurts about every fifteen minutes. "In order to stop the bleeding from the nose," a weak solution of adrenalin was "snuffed" up into the nose and adrenalin gauze packing inserted. Following this meddlesome treatment, and three hours after the injury, the patient was brought to the hospital in the ambulance; the expectant palliative treatment alone was indicated and the patient was making such an excellent recovery that he was permitted to sit out of bed on the sixth day after the injury. On the eighth day, however, the patient suddenly had a general convulsive seizure and remained unconscious; the temperature ascended to 104.8 and a lumbar puncture removed cloudy cerebrospinal fluid containing numerous streptococci, so that no cranial operation for drainage was advisable. An autopsy, twenty hours later, confirmed the röntgenograms and disclosed a purulent meningitis and meningo-encephalitis of both frontal lobes; much free pus (streptococci and staphylococci) was found about the fractures of the cribriform plate of the frontal bone—the channel for the infection.

These patients in shock may or may not be unconscious. A cold, clammy skin, with pallor of greater or less degree, is usually present; a subnormal temperature; the pulse-rate is usually 130 plus, being of a weak, thready, and irregular character in the severe cases, while the respiration-rate is between 30 and 40, shallow, and at times scarcely perceptible; the blood-pressure may be 100—rarely below (Fig. 10). This period of severe initial shock rarely lasts longer than 12 hours; usually it is about 6 hours after the cranial injury before the patient shows definite signs of improvement: a lowering of the pulse- and respiration-rates, while the temperature and blood-pressure rise. If the patient is unable to survive an extreme condition of shock, then an early pulmonary edema occurs—moist râles throughout both lungs, and the patient quickly succumbs—within 24 hours, and usually within 6 hours after the injury.

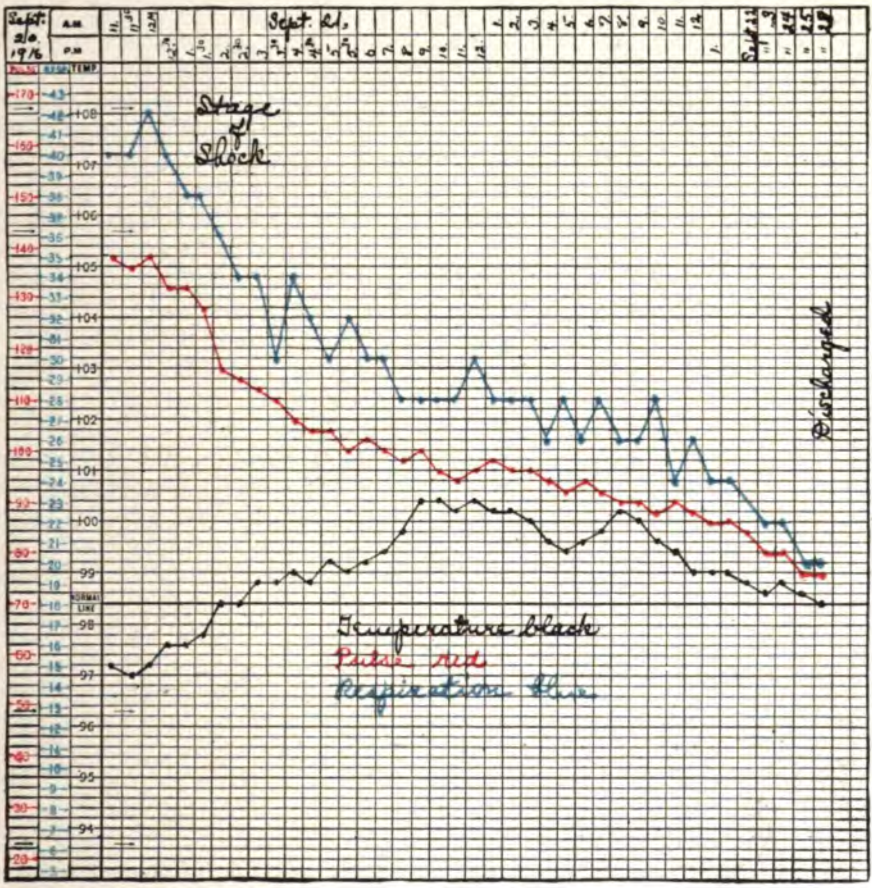

FIG. 10.—No. 624.—Alice, eighteen years. Clinical chart presenting the typical temperature, pulse and respiration syndrome of an uncomplicated condition of shock following a cranial injury with multiple fractures of the vault and base of the skull. Uneventful recovery from the shock within six hours, and as no signs of a marked increase of the intracranial pressure were then disclosed by the ophthalmoscope and the spinal mercurial manometer (although the cerebrospinal fluid was blood-tinged), this patient could be discharged from the hospital on the eighth day after the injury. Last examination (September 6, 1919)—twenty-four months after the cranial injury: no complaints; physical examination—negative.

ondition of severe shock can usually be differentiated from that of the terminal stage of medullary edema and collapse by the clinical history and the temperature chart. In these patients of medullary edema, the pulse- and respiration-rates have been low for a period of minutes, and even hours, following the cranial injury—that is, the signs of medullary compression—and then, after the lowest level of the pulse- and respiration-rates for the patient has been reached, such as 46 and 12 respectively, there is a rapid rise, so that within two hours the pulse may be 70 and the respiration 24, and within six hours the pulse 110 and the respiration 30, and two hours later even 140 and 40 respectively, and at the same time the temperature quickly rises to 105 and above, while the blood-pressure descends to 110 and lower—the condition of medullary edema, and not of shock or delayed shock, has now unquestionably occurred. Medullary edema and collapse is the bane of brain lesions, traumatic and otherwise, and it cannot be feared too much, since it is always the forerunner of death in these patients. In the most extreme cases, and particularly in the subtentorial lesions, the condition of medullary edema may occur within a few minutes after the cranial injury, and the patient be dead before a competent examination in a hospital is possible; it is in these patients that a post-mortem examination is essential, in order to state whether extreme shock or medullary collapse was the immediate cause of death.

2 ~~1~~. *Temperature.*—As an index of the general condition of the patient following a cranial injury, the temperature is helpful, and especially so during the two extreme and most dangerous periods—the period of initial shock, when the temperature is subnormal, and then the terminal stage of acute medullary edema, when the temperature ascends rapidly to 105 and higher. Naturally, the other clinical signs should be considered with the temperature, but together they form a picture which is usually a most characteristic and typical one of the period in which the patient having a brain injury happens to be. It is extremely rare for these patients to have a normal temperature, and, if the condition of shock is excluded, there is almost always a temperature of 100 to 102, varying as the condition of the patient changes. Temperature in itself, however, is no indication of the degree of intracranial pressure, unless a medullary edema has been produced—and then it is too late to utilize the temperature findings in the treatment of the patient, as these patients in medullary edema all die, treatment or no treatment. Injuries of the base of the brain and supposedly "upsetting" the basal ganglia would cause an early rise of temperature, and are not necessarily associated with the other clinical signs of cerebral injury.

3. *Pulse.*—Owing to the usual presence of shock of varying degree, the pulse-rate following a brain injury is frequently 120 and higher, and this may continue for several hours, until the shock gradually disappears; the pulse will then become lowered to its normal rate, and may become much less; if it should reach 60 or lower, a definite degree of intracranial pressure is usually present. However, the normal pulse-rate of the patient before the injury should, if possible, always be ascertained. The pulse-rate, unfortunately, is not an accurate or an early means of determining the severity of the intracranial lesion; ordinarily, the greater the intracranial pressure and, therefore, the less the blood supply to the medulla, the slower the pulse-rate; but the resistance of the medulla to slight changes in its circula-

tion varies so much in different individuals that it is possible to have a high degree of intracranial pressure and yet a pulse-rate remaining between 70 and 80 for a period of hours. Finally, the regulatory mechanism of the medulla may become affected, and then the signs of medullary compression advance unusually rapidly, leading to the quick death of the patient. I have had several patients in whom the pulse-rate remained between 70 and 80, and yet the ophthalmoscope revealed marked signs of high intracranial pressure; at operation, the high pressure was confirmed, being usually due to a markedly swollen edematous brain, with numerous punctate hemorrhages in its cortex and the subdural cerebrospinal fluid being blood-tinged.

If the pulse-rate, however, becomes lowered to 60, and especially to 50, we have an excellent though rather late danger signal, which should always be heeded as an evidence of medullary compression (Fig. 11). Any further lowering of the pulse-rate is usually associated with an irregular respiration-rate of the Cheyne-Stokes type, and then the prognosis becomes very poor indeed.

As the pulse-rate descends, the character of the pulse itself becomes full, strong, and well sustained—apparently an excellent pulse. It is, however, only the attempt of the circulatory mechanism to overcome the partial anemia of the medulla, due to the increased intracranial pressure, and thus to force blood into it. If this condition remains for a variable length of time, signs of medullary edema and finally collapse—rapid, shallow pulse, and quick, irregular respirations—may occur at any moment, and then death is merely a matter of hours.

Many of the most severely injured and moribund patients, even at the first examination within one hour after the head injury, have a pulse-rate over 120, either of pure shock or of shock associated with the early stages of medullary edema. If the rapid and shallow pulse-rate, associated with a subnormal temperature, is due simply to shock, then there is a definite chance of recovery, as shown by the gradual disappearance of the signs of shock and the lowering of the pulse-rate; if, on the contrary, medullary edema is progressing, then, together with the elevated temperature, the pulse-rate remains high and gradually ascends until it is no longer obtainable before the death of the patient. Naturally, all of these patients should be treated expectantly and for the condition of shock, in the hope that the medullary edema is only a temporary complication. It must be admitted, however, that once a true medullary edema does occur, then the pulse-rate rapidly ascends, becomes more and more shallow, and I have yet to see such a patient recover. Any operative procedure at this stage of extreme shock and medullary edema and collapse merely hastens the exitus of the patient.

4. *Respiration.*—The respiration, like the pulse-rate, is affected by shock, and may exceed 40 for an hour or more immediately after the injury; its rate, however, becomes the normal 20 to 24 much more quickly, and remains normal unless the intracranial pressure becomes so high as to cause the definite medullary signs of a Cheyne-Stokes type of breathing. In these patients, the period of apnea or non-breathing may exceed 40 seconds. It seems that the pulse is more easily affected by intracranial pressure than the respiration, and when the latter is influenced a medullary edema and collapse may occur at any moment (Fig. 12).

An early medullary edema of rapid onset, and associated or not with severe shock, may prevent the respiration-rate, just as it does the pulse-rate, from being lowered, so that from the time of the first examination immediately after the cranial injury until the death of the patient, the respiration is continuously shallow and of a rate higher than 40. These are the so-called moribund patients, who die usually within six hours after the injury. In many of these patients the brain injury is associated with internal injuries of the abdomen and of the chest and thus lessening still more the chances of recovery. The usual presence, however, of an extreme degree of shock in these patients always makes the vigorous treatment of the shock most imperative.

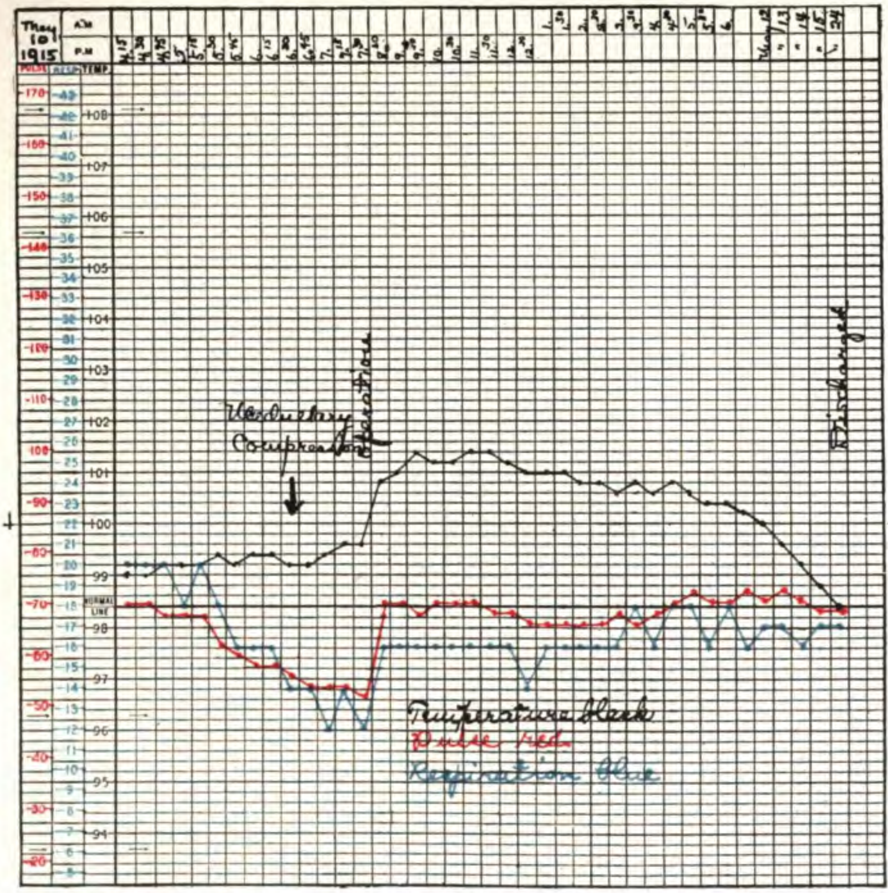

FIG. 11.—No. 312.—William, forty-two years. Clinical chart representing the rapid onset of acute medullary compression within three hours following a severe cranial injury but not associated with a fracture of the skull. Signs of an increasing intracranial pressure were also disclosed by the ophthalmoscope and the spinal mercurial manometer, although the cerebrospinal fluid at lumbar puncture was clear. At the operation of right subtemporal decompression, a "wet" edematous cortex was revealed under high pressure, so that upon incising the dura the clear cerebrospinal fluid spurted to a height of ten inches; no hemorrhage observed. Uneventful recovery. Last examination (December 14, 1918) — nineteen months after injury: no complaints; physical examination—negative.

5. *Blood-pressure.*—It has been most interesti ients having

brain injuries to record the influence of intracranial pressure upon the general blood-pressure. It was surprising to ascertain that rarely was the blood-pressure forced beyond 160, and then only in the patients showing early signs of medullary compression; in these patients the blood-pressure might ascend to 200, with a pulse-rate of 50 and below, and a Cheyne-Stokes respiration, showing an extreme degree of intracranial pressure and one that could not be long sustained by the medulla. These were the patients allowed to wait a number of hours before the operation of cranial decompression was performed, and their recovery was most doubtful. The operative mortality is very high for patients that have been allowed to reach this extreme degree of medullary compression; signs of a medullary edema and collapse—high pulse-rate of a shallow, poorly sustained char-

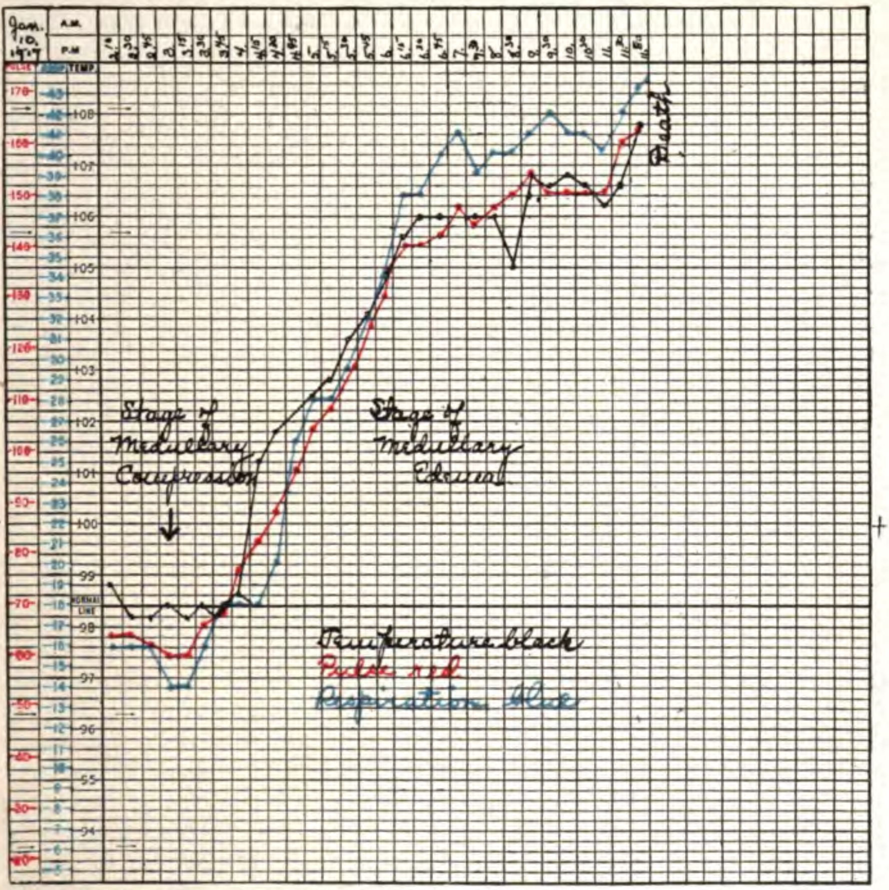

FIG. 12.—No. 892.—Arthur. Clinical chart illustrating the temperature, pulse and respiration syndrome of the stage of medullary compression merging into that of acute medullary edema with the usual result—the death of the patient. Upon admission to the hospital, the fundi were negative but the spinal mercurial manometer registered a pressure of 20 mm.; while waiting for the operating room to be prepared, the temperature, pulse- and respiration-rates began to ascend rapidly within one hour after admission to the hospital, showing that the stage of medullary compression had advanced into that of medullary edema, and therefore it was too late to perform the operation of cranial decompression in order to be of benefit to the patient. The autopsy revealed a subdural hemorrhage with extreme cerebral edema, but no fracture of the skull.

acter, irregular shallow respirations, and a blood-pressure of 100 and even less—usually followed within several hours until death, operation or no operation.

A number of years ago, venesection was considered advisable to lower this increase of the general blood-pressure associated with medullary compression; von Bergman and Leonard Hill thought favorably of it. However, it is no longer employed, because it is now known that this increase in the general blood-pressure is the attempt of the natural vasomotor mechanism to force blood into the intracranial chambers, and thus into the medulla, by overcoming the increased intracranial pressure. To bleed a patient, therefore, even in the very mild cases, is an exceedingly dangerous procedure, of no real value, and is always contraindicated.

At best, the general blood-pressure is a very crude method of estimating the intracranial status of pressure. Besides, the factor of initial shock, which is almost always present in varying degree for several hours at least, tends to conceal the true intracranial condition. The high intracranial pressure necessary to raise the general blood-pressure to any appreciable extent must reach a very dangerous stage of compression in these acute cases before its effects can be observed in an increased blood-pressure. Particularly is this true of a cerebral and supratentorial increase of pressure, whereas a cerebellar and subtentorial increase of pressure produces an almost immediate rise in the blood-pressure. Naturally, these latter conditions are most serious ones, and rarely do these patients survive, operation or no operation. I feel, therefore, that the blood-pressure as an important aid in the treatment of brain injuries has been very much overemphasized. I have had the blood-pressure ascertained in all of my patients, but it has rarely been of any material assistance as an indication of the status of the patient, except as an additional diagnostic aid in the subtentorial lesions. It should be remembered that brain injuries are acute conditions associated always with shock, whereas brain tumors are usually of slow growth, and not associated with the masking factor of shock. It has been a most interesting study to observe the rhythmical variations of blood-pressure synchronous with the variations of temperature, pulse- and respiration-rate, a lowered blood-pressure (100–120) during the period of initial shock, associated with a subnormal temperature and increased pulse- and respiration-rate. As the shock lessened, then the blood-pressure slowly ascended (130–140) with the temperature, while the pulse- and respiration-rate descended. If the intracranial pressure became very high, then the blood-pressure continued to ascend slowly (140–160), the temperature remaining above normal (101–103), and the pulse- and respiration-rate dropped to 60 and 16, respectively, and even lower. If medullary edema should be permitted to occur, then the blood-pressure descended rapidly to 100, and even lower, while the temperature rose to 105 plus, and the pulse- and respiration-rate to 150 and 40 plus, respectively, to be followed within several hours by the exitus of the patient.

6. *Paralysis.*—The condition of central or cortical paralysis, and usually only a paresis or weakness of the extremities, does not in itself indicate the necessity of a cranial operation. If there are no marked signs of in-

creased intracranial pressure, and a depressed fracture of the skull cannot be ascertained, then the expectant palliative treatment should be used in the belief that the paralysis is only of temporary duration, and therefore due to localized edema of the motor cortex with or without being associated with a small laceration of the adjacent cortex. If the definite signs of an increased intracranial pressure appear, or the paralysis continues for a period of days, then an exploratory decompression and drainage would be indicated over the hemisphere involved. Naturally, a large cortical laceration through either motor area means a permanent damage to the cells destroyed, there being no cell regeneration possible; but there is always associated with these extensive lacerations and contusions of the cerebral cortex a profuse localized cerebral edema, so that the cortical nerve cells surrounding the lacerations are merely compressed and functionally impaired by the edema, which is usually of such an amount as to increase the intracranial pressure. Therefore, in these patients, as a result of the signs of this increased intracranial pressure, the operation of subtemporal decompression and drainage is advisable, and thus an early recovery of function of the paralyzed extremities is facilitated. The milder degrees of cortical laceration and localized edema do not produce the definite signs of a marked increase of the intracranial pressure, and therefore no cranial operation is necessary, the natural means of absorption alone being sufficient.

Unless the fracture of the skull is a depressed one, over either motor tract, so that the underlying cortex is compressed, and even lacerated, or there is a large extradural hemorrhage (due to a rupture of the middle meningeal artery), and, less frequently, a subdural clot overlying the same area, or a general or localized edema of the motor area, with or without a definite cortical laceration, it is rare for a fracture of the skull to produce paralysis of the extremities; especially is this true of fractures of the base.

Paralyses of the cranial nerves and particularly those controlling the movements of the eyeball—the third (oculi motorius), the fourth (patheticus), and the sixth (abducens), and also of the seventh (facialis)—are fairly common in basal fractures, resulting in ptosis, strabismus, and facial paralysis. If the nerves have been severed, then a permanent paralysis results, but most frequently the paralysis is of temporary duration only, and it fades away after the local pressure of hemorrhage or edema has been removed by absorption; this is particularly true of the facial nerve in its narrow bony canal in the aqueduct of Fallopius—its edematous compression producing the peripheral type of facial paralysis. The other cranial nerves occasionally affected are the first (olfactorius), the fifth (trigeminus), and, more frequently, the eighth (auditorius). The second (opticus) and the other cranial nerves are rarely primarily affected.

A temporary motor aphasia frequently results from a subdural and supracortical hemorrhage overlying the motor speech area, and from a partial laceration of the cortex adjacent to the left third frontal convolution posteriorly; it is usually merely a paraphasia, a pure motor aphasia being most rare.

The absence of paralysis, therefore, is of little significance of the seriousness of brain injuries. The advisability of an operation or not is rarely lependent upon the presence or the absence of paralysis. In many patients, ind possibly the majority of those having cortical paralyses, the paralysis is due to a localized edema, or even a definite laceration of the motor cortex itself—not a complete destruction of it, but a partial tear, and associated with the resultant cortical edema; thus, a sufficient cause for a distinct paralysis of the hemiplegic type, with or without a marked increase of the intracranial pressure. It is rare for the hemiparesis to be a permanent one, on account of the absorption of the cerebral edema about the cortical laceration, which extends throughout the motor tracts in only very infrequent cases. Naturally, no operation would be indicated for a laceration of the brain unless it was associated with a definite increase of intracranial pressure, which may or may not be present. The frequent statement, therefore, that paralysis is an indication for operative interference in brain injuries cannot be credited. If there is no increased intracranial pressure, due to hemorrhage or to cerebral edema, surely no operation will be of any assistance to the patient.

The great frequency of cortical lacerations and contusions of the "*contre-coup*" type is very impressive. The anterior and inferior surfaces of the frontal lobes are usually the areas affected, due undoubtedly to the direct contact of the cranial trauma being usually in the region of the posterior occipital bone. The tips of the temporo-sphenoidal lobes are lacerated by "*contre-coup*" next in the order of frequency. As both the frontal and the temporo-sphenoidal lobes are comparatively "silent" areas of the brain, it will thus be seen that the condition is rarely diagnosed; besides, the treatment remains the same, laceration or no laceration of the cerebral cortex.

In this connection, the possibility of a laceration of the pyramidal tract fibres in their course from the cortex down through the internal capsule to the pons and medulla should be remembered; fortunately this complication rarely occurs, and the same may be said regarding an active hemorrhage into the ventricles and internal capsules following cranial trauma, and thus producing pyramidal tract compression; their possibility in elderly patients is theoretically always to be feared. The expectant palliative treatment would here be indicated, although in an acute traumatic ventricular hemorrhage of sufficient size to produce an increased intracranial pressure, an early subtemporal decompression, with drainage of the ventricle, might be indicated in selected patients; I have operated upon four patients, with two recoveries.

7. *Impaired Sensation.*—It is rare for areas of hypesthesia and anesthesia to be present, and most rare for areas of hyperesthesia to exist in these traumatic cranial patients. They do occur, however, if a large extradural or subdural hemorrhage exerts a pressure over the post-Rolandic cortical area sufficient to lessen its sensitiveness to afferent impulses. Usually, however, merely a mild hypesthesia results; an astereognosis may or may not be present. This cortical impairment of sensation of one-half of the body is usually associated with a definite hemiplegia of the same side, on account of the extension of the hemorrhagic clot forward beyond the fissure of Rolando, and thus over the motor area. Subcortical lacerations or hemorrhage may affect the afferent sensory pyramidal tract fibres just as they

do the efferent motor fibres of these tracts, and the comment is the same as in the preceding paragraph upon paralysis. Traumatic lesions of the internal capsular fibres rarely occur without the definite association of motor and sensory impairments of the opposite side of the body, similar to apoplexy of the internal capsular type (the usual form). Hypesthesia and even anesthesia of the ipsolateral half of the face may result from compression or direct injury to the fifth cranial nerve (trigeminus or trifacial); rarely is this impairment a permanent one, being usually due to an edema of the nerve itself, following a fracture of the adjacent portion of the petrous bone.

8. *Unconsciousness.*—Prolonged total loss of consciousness in patients having brain injuries, with or without a fracture of the skull, usually indicates a high degree of intracranial pressure, due to hemorrhage or edema, or an extensive laceration and destruction of the brain substance itself, with comparatively small amount of associated hemorrhage and edema. Loss of consciousness, however, is not necessarily associated with a high intracranial pressure, nor does a high intracranial pressure always produce a loss of consciousness. Some of the patients in this series of brain injuries, having the highest intracranial pressure (sufficient to produce the early signs of a beginning medullary compression), were called only "unusually drowsy" and "stuporous," and were easily aroused by supra-orbital pressure, by pricking the skin, or even by calling the patient's name.

In cases of slowly progressing hemorrhage and of edema intracranially, as revealed by the ophthalmoscope, lumbar puncture and confirmed by operation, the various stages of "feeling of tiredness," then drowsiness, stupor, coma, and finally total unconsciousness, may be observed within a period of several hours. It is not a very unusual occurrence to have such patients walk into the hospital with the complaint of "throbbing" in the head, and then pass gradually through these stages to total unconsciousness. The vast majority of patients, however, following a brain injury, with or without a fracture of the skull, are more or less comatose, and it is of the greatest importance to observe whether the degree of coma lessens or increases, according to variations of the intracranial pressure.

9. *Restlessness.*—Instead of being drowsy and stuporous, a large number of the patients having brain injuries, with or without a fracture of the skull, are in the irritative stage of the condition—that is, owing to the sudden oozing of supracortical blood and to the edema of the cerebral cortex itself, immediately following the brain injury, there is a definite irritation of the underlying cerebral cortex—merely a lessened emotional control in the very mild cases, to the extreme degrees of restlessness, mental excitement, and even acute mania in the more severe cases. Naturally, this violent "threshing about" in the bed and the vigorous muscular exertions should be prevented by every means possible, as the danger of increasing the intracranial lesion of hemorrhage and edema is very great. Besides the routine application of the ice helmet (surrounding the entire head and strapped to the head), triple bromides and chloral in large doses should be given; morphia is frequently effective. Since the adoption of the routine use of the ophthalmoscope and of the spinal mercurial manometer for estimating the intracranial pressure, there is little or no danger of morphia masking the true

intracranial condition. Formerly, it was advisable to restrict the administration of morphia, for fear that the intracranial condition might be concealed, and the proper treatment thereby so delayed until the condition of the patient became most serious. This was perfectly true formerly, but since it has become more and more recognized that no cranial operation is indicated in these patients unless there are definite signs of an increased intracranial pressure (as can very easily be determined by the ophthalmoscope and by the spinal mercurial manometer), there should be no longer any hesitancy in administering morphia in sufficient quantity to control the patient.

10. *Convulsive Seizures.*—Spasmodic twitchings, and even convulsions, usually of the localized Jacksonian type, occur very frequently as the result of an acute cortical irritation, due to the presence of subarachnoid and subpial (cortical) hemorrhages and edema; occasionally subdural clots produce them, as confirmed by operation. Convulsive seizures are very infrequent in the other forms of intracranial pressure and hemorrhage. Acute depressed fractures of the vault rarely cause sufficient cortical irritation to produce convulsions until months, and even years, later, and then only when associated with a chronic cerebral edema to the extent of increasing the intracranial pressure.

The comparative infrequency of convulsions associated with acute brain injuries is very singular. In this series of 487 patients having acute brain injuries, in only 27 of them were convulsions or even localized twitchings to be observed. At the operations upon 155 of these patients as well as at the autopsies upon the 69 non-operated patients who died, the presence of subdural and cortical hemorrhage and of cortical edema was very frequently demonstrated as a possible cause of convulsive seizures and twitchings, and yet convulsions rarely occurred. Possibly the cortical nerve cells in these acute cases were less sensitive to local irritation, being "benumbed," as it were, by the shock and acute cerebral edema, and thus the "explosive" reaction producing convulsions was inhibited—merely a theoretical explanation.

Just why a small number of these patients develop epilepsy later is, in my opinion, not so much a question of possible cortical adhesions, depressions of the vault or foreign body spicules, but, rather, in addition to these factors, a condition depending upon the permanency of the cerebral edema producing a chronic increase of the intracranial pressure, and especially in patients of lessened stability nervously. I have examined a large number of these post-traumatic epileptics, some of them having had but two or three convulsive seizures, and yet they all had chronic "wet," swollen, edematous brains, causing a definite increase of the intracranial pressure. Many of these patients did not have cortical adhesions, to be disclosed either at operation or at autopsy. In this connection, it must always be remembered that frequent convulsions will eventually produce a "wet" edematous condition of the brain, so that any increase of the intracranial pressure in many of the chronic patients is a secondary one, resulting from the convulsions rather than being the primary cause of the convulsions. A method of determining whether the increased intracranial pressure in these post-traumatic epileptics is primary or secondary to the convulsions, is to ascertain the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer, then by the vigorous use of triple bromides, luminal, etc., to prevent the convulsive seizures from occurring for a period of at least one month, and, better, six weeks. If the pressure as registered by the spinal mercurial manometer is practically the same as at the preceding examination immediately following the last convulsion, then the increased intracranial pressure is primary as concerns the convulsive seizures; whereas, if the measurement of the pressure of the cerebrospinal fluid is now markedly lowered to almost normal, then it may be concluded that the increased intracranial pressure is secondary to the convulsions—that is, the convulsions are causing the chronic cerebral edema, rather than the cerebral edema being a factor in producing the convulsions. This test is an important one in regard to the advisability of any operative procedure to relieve the increased intracranial pressure in these patients; and naturally no cranial operation of decompression can be considered for those patients in whom the increased intracranial pressure is secondary to the convulsions, and is therefore not one of the primary factors of the convulsions. To operate upon the former group of patients, in whom the intracranial pressure is secondary to the convulsions, and then to expect a subtemporal decompression to be of benefit, is absurd, and this neglect to differentiate these two types of post-traumatic epilepsies has in reality discredited cranial surgery in the treatment of selected cases of post-traumatic epilepsy. The surgical treatment of convulsive seizures in these patients is notoriously bad, and it will remain so unless greater care is used to ascertain which patients are amenable to operative treatment—and only a very small percentage of them are; and then, upon these early selected patients having a primary increased intracranial pressure, a simple subtemporal decompression will occasionally be of permanent benefit. At best, however, the surgical treatment of these selected patients should only be used after the most careful study and consideration, and in only the very early cases.

It would seem to be a fact that the brain cells of certain neurotic patients are more unstable than those cells of the more stable and phlegmatic types of patients, so that a less active cortical irritant is necessary to cause convulsions in the former class than in the latter class of patients. Alcohol, as a factor, must always be considered, because it increases the cortical irritability, and renders the cells less stable; it alone may even produce convulsive twitchings and seizures.

11. *Reflexes.*—The presence of shock in these acute cases is an important factor in influencing the activity of the superficial and deep reflexes; in the mild degrees of shock, the skin-reflexes cannot be elicited while the deep reflexes are present, and in the extreme condition of shock they may both be entirely absent. As the patient recovers from the shock, first the tendon-reflexes return, and then the skin-reflexes. In those patients who emerge from the condition of shock to enter the stage of intracranial pressure, the abdominal reflexes will frequently appear depressed on the side opposite to the cerebral hemisphere more compressed and the cerebral lesion, and at the same time it is usually possible to obtain a definite Chaddock reflex; but the Babinski reflex is absent, and reappears only later, especially if the abdominal reflex cannot then be elicited. It may be that the Chad-

dock method of eliciting the dorsal extension of the great toe is possibly a more delicate one than the Babinski method, as it is in many patients its forerunner.

These reflexes usually become more and more active, until they are distinctly exaggerated, and if either pyramidal tract is compressed or injured intracranially, then the definite signs of such involvement are to be recognized on the opposite side of the body by the patellar- and ankle-clonus, the extensor flexion of the large toe upon plantar stroking (Babinski's sign), and markedly increased tendon-reflexes of the arm and leg, whereas, at the same time the abdominal skin-reflexes are usually lessened or even abolished. It is, however, rare for these signs to be unilateral alone, unless in patients with lesions affecting only one side of the cortex and especially in the presence of a large unilateral extradural hemorrhage. In the majority of cases of brain injuries, both sides of the brain have been so damaged and impaired by the general increase of the intracranial pressure by hemorrhage and cerebral edema, that there is a marked exaggeration of the reflexes of both sides of the body, and in many patients a bilateral extensor reflex of the toes; this latter sign may last but a few hours in the mild cases, showing that no extensive damage has occurred to the pyramidal tracts. However, it is a very reliable sign, and its presence is always very significant. In patients where one side of the cortex has been damaged more than the other side, the clonus and the Babinski sign will persist on the side of the body opposite the more damaged cerebral hemisphere, and gradually fade away upon the side of the body opposite the less damaged cerebral cortex.

Besides the tendon-reflexes in the arms as a means of ascertaining a lesion of the pyramidal tract, a sign similar to the Babinski reflex of the foot is frequently useful in the hand—the so-called Hoffman sign; by pinching sharply the end of the forefinger, the terminal phalanx of the thumb flexes briskly if there is a definite lesion of the pyramidal tract of the opposite side; this sign, however, is less frequently elicited than the Babinski sign.

Increased intracranial pressure due to a simple edema alone is sufficient to produce these signs of pyramidal tract impairment, and the signs will persist as long as the edematous condition remains—in the mild cases for several days, and in the more severe cases for two weeks, and even much longer. In a number of patients having brain injuries of moderate severity, exaggerated reflexes, and even a bilateral Babinski reflex, may often persist longer than three months after the dáte of the injury, associated usually with a general nervous instability characterized by restlessness, irritability, and emotionalism of the extreme type, the patient being much depressed, with frequent crying spells, and very easily angered.

Thus, in patients having high intracranial pressure, careful and most thorough neurological examinations are essential in localizing accurately, whenever possible, the exact site of the intracranial hemorrhage. Naturally, the hemorrhage should be drained, if possbile, but it is rather infrequent in these patients that this can be done. It is usually more important in the treatment to offset the intracranial pressure of the hemorrhage by a simple operative procedure of decompression and drainage in the subtemporal area, rather than to attempt a removal of the hemorrhage itself by the more extensive operation of an osteoplastic "flap" over a more highly developed area of the cerebral cortex. If the intracranial pressure is high, then the underlying cortex may be permanently damaged by its protrusion either through the "bone flap" opening or through the common small trephine opening—a most dangerous and inadequate procedure.

12. *Pupillary Changes.*—Owing to the presence of initial shock of varying degree in almost all of these patients having brain injuries, the pupils are usually slightly enlarged when associated with a mild condition of shock, and widely dilated in the more severe stages of shock; the reaction to light is correspondingly sluggish. If the patient is also unconscious, then the pupillary dilatation and the sluggish reaction to light are always increased. The presence of alcoholism is also a factor in accentuating this change. As the patient recovers from the condition of shock, and regains consciousness (if that has been prolonged), the pupils usually return to normal size and light reaction, and remain so unless there is present a definite increase of the intracranial pressure. In this latter condition the pupils may remain slightly enlarged, with sluggish reaction to light.

A marked constriction and "pin-point" pupils result from a cortical irritation of a supracortical hemorrhage and mild cortical edema, producing the "irritative" stage of pupillary contraction. If this cortical irritant of hemorrhage and edema increases until the supracortical and cortical pressure becomes high enough to compress the cortical nerve cells, then the pupillary narrowing of the "irritative" stage yields to the pupillary dilatation of the "paralytic" stage, due to the compression of the cerebral cortex, and thus dilated pupils with sluggish light reaction appear. This pupillary phenomenon can be frequently observed in patients having brain injuries in the progress of a supracortical hemorrhage and cerebral edema. If morphia has been administered to control the patient, or if the patient is an addict, then careful and accurate ophthalmoscopic examinations can still be made, while the accuracy of the spinal mercurial manometer in registering the pressure of the cerebrospinal fluid is in no way impaired.

Inequality of the pupils persisting after the initial stage of shock may be due to a direct impairment of the third cranial nerve (oculi motorius), the cervical sympathetic branches, or, and most frequently in these traumatic patients, to the influence of the presence of hemorrhage and edema upon the cerebral cortex of either hemisphere. The third cranial nerve is less easily impaired than the sixth (abducens), but it may be temporarily compressed or even severed in rare cases, producing an enlargement of the pupil (owing to the unopposed action of pupillary dilatation of the cervical sympathetic) and a weakness particularly of the internal rectus muscle of the eye, and thus an outward rotation of the eyeball (unilateral divergent strabismus), owing to the unopposed action of the external rectus muscle (supplied by the sixth nerve). Unilateral constriction of the pupil may be due to an impairment of the homolateral cervical sympathetic system and its connecting spinal branches in the lower cervical and upper dorsal nerve

thus permitting the unopposed constricting action of the third nerve.

be remembered that, in the irritative lesions of the third nerve and the cervical sympathetic, their normal action is usually increased, so that an irritative lesion of the third nerve produces a pupillary constriction, whereas a similar lesion of the cervical sympathetic causes a dilatation of the homolateral pupil. (This latter pupillary phenomenon can be strikingly demonstrated by pinching the skin at the base of the neck over the outer third of the clavicle, when an immediate enlargement of the homolateral pupil will be easily observed.)

The usual cause of pupillary inequality in these patients having brain injuries after the acute stage of shock has been passed is the pupillary constriction of an "irritative" lesion of the cerebral cortex of the homolateral hemisphere—that is, if the supracortical hemorrhage or cerebral edema is of mild degree, and only sufficient to be an irritant of the cerebral cortex, then the homolateral pupil is narrowed; whereas, if the pressure of the supracortical hemorrhage and edema increases until it compresses the underlying cerebral cortex, then the homolateral pupil becomes enlarged, and in the severe cases of cerebral compression the pupillary dilatation may be the maximum possible.

These pupillary changes, however, are of the greatest significance only when the entire clinical picture is considered. In the absence of the more accurate signs of increased intracranial pressure, as elicited by the ophthalmoscope and the spinal mercurial manometer, then a pupillary "paralytic" dilatation is of interest, but of little importance in the treatment of the patient; and, conversely, a pupillary "irritative" constriction, in the absence of other signs of cortical irritation, such as extreme restlessness and even localized convulsive seizures, is an interesting observation, but not in itself of sufficient importance to warrant a marked change in the treatment of the patient. The expectant palliative treatment of such a patient could never be abandoned for the operative treatment merely on account of these pupillary changes alone. They should always be noted, however, in order to complete the clinical picture, and thus to aid in the diagnosis of the actual intracranial condition as accurately as possible.

13. *Urinary Findings.*—It is very important that a routine examination of the urine should be made as early as possible in each patient having a brain injury. The associated unconsciousness may be due primarily to a cardio-renal disease, which must be eliminated as a possibility. So common are the complications of cardio-renal and arteriosclerotic diseases in these adult patients of middle age and older, that it is essential to ascertain their presence or absence in order to vary the treatment accordingly, and also to insure the probable prognosis—always a most difficult task, and one which must be most guarded. The effect of chronic alcoholism can also be estimated. The great danger in these patients having nephritic complications is the acute onset of cerebral edema—a "wet," edematous brain to which they are all especially liable, even if the cranial injury is apparently a trivial one. In some of these patients it would seem that mere confinement in bed for 24 hours increased their susceptibility to an acute cerebral edema, being particularly so in the development of delirium tremens in chronic alcoholics.

Brain injuries affecting the pituitary body, and especially basal fractures extending into the middle fossa and across the sella turcica, will frequently cause sugar to be temporarily excreted in the urine, as disclosed by the Fehling reaction and the other routine tests. This appearance of sugar rarely occurs before six hours after the cranial injury, and, in this series of patients, it has never been demonstrated to persist longer than 36 hours—that is, the pituitary lesion is thus merely an irritative one affecting the anterior lobe, rather than a definite lesion or destruction of its cells themselves.

14. *Ophthalmoscopic Findings.*—The presence or not of "choked disks" as *the* sign of an increased intracranial pressure has possibly retarded the recognition of the earlier signs of an increased intracranial pressure more than any other factor. It is still commonly believed that unless a "choked disk"—a papilledema—of 2 diopters or more, is present, then there is no increase of the intracranial pressure, and thus overlooking the earlier degrees of intracranial pressure such as a dilatation of the retinal veins, an edematous blurring of the margins and an edema of the halves of the optic disks themselves, but not of a measurable swelling, or, if so, then merely a papilledema of one or two diopters, these being the preliminary stages of the condition of "choked disks," which is the advanced result of very high intracranial pressure. Merely because this extreme condition of "choked disks" is not present does not mean that there is not a definite increase of the intracranial pressure, and of sufficient degree to produce the dangerous complication of medullary compression, and even edema itself. It is these early stages of the fundal signs of an increased intracranial pressure which have been overlooked in the past, but which can now be demonstrated and also confirmed by means of the spinal mercurial manometer at lumbar puncture.

Besides the lowered pulse- and respiration-rates, which are comparatively crude signs of intracranial pressure, and if of the irregular Cheyne-Stokes type, then most late signs of extreme intracranial pressure with its resulting medullary compression, the two most valuable procedures for determining a definite increase of the intracranial pressure are the examinations of the fundi of the eyes with the ophthalmoscope, and the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer.

Although it is rare for a measurable papilledema and "choked disks" to occur in these patients having traumatic intracranial lesions, with and without a fracture of the skull, yet the earlier, and, therefore, milder degrees of an edema of the optic disks should be most carefully "watched for" with the ophthalmoscope, as being one of the accurate signs of the presence or not of a definite increase of the intracranial pressure. The ophthalmoscope (Fig. 13), and especially the "direct" method, using an electric light, is a most valuable aid in the diagnosis of intracranial lesions. Proficiency in the use of this instrument is essential to accurate diagnosis; one should practice first with normal eyes, so that any abnormal dilatation of the retinal veins and edematous changes of the optic disks may quickly be noted. The proficient use of this "direct" method in the examination of the fundus is not only simpler and easier to acquire than the old "indirect" method of reflected light and an interposed lens, but it is much more

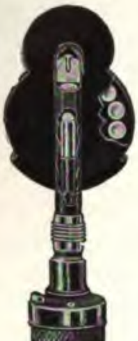

FIG. 13.—A simple electric ophthalmoscope (one-half the actual size) for the determination of the presence or not of an increased intracranial pressure, and its degree, by the direct method. Two small electric batteries are enclosed in the cylindrical metallic handle so that this useful instrument may be easily carried in a coat pocket—its weight being less than one-half pound. Its simplicity and durability minimize the necessity of repairs from careless handling. The very early stages of papilledema, as well as the advanced degrees of "choked disks" can be accurately measured without the necessity of artificial light or the interposition of lenses, as required by the former indirect method of ophthalmoscopic examinations.

accurate in revealing the minute details and changes in the fundus of the eye. With the "indirect" method, it is sometimes difficult to distinguish slight pathological changes occurring in the fundus.

In occasional cases of severe concussion, with and without a fracture of the skull, it is possible for the opthalmoscope to reveal a slight dilatation of the retinal veins, that is, a moderate degree of increased intracranial pressure. In mild cases, the intracranial pressure does not advance beyond this height, which produces merely a dilatation of the retinal vessels, and in many of the cases of simple concussion not even a dilatation of the retinal veins results.

It is in those patients, however, having not only a dilatation of the retinal veins, but the added blurring and haziness of edematous optic disks, that we should be careful to make repeated ophthalmoscopic examinations of the fundi in order to ascertain the earliest signs of a still increasing intracranial pressure—whether it is due to a simple edema of a "swollen" brain or to a hemorrhage. The signs of a still increasing intracranial pressure beyond a dilatation of the retinal veins are, first, an edematous blurring and obscuration of the nasal margin of the optic disk (Fig. 14), then a similar haziness of its temporal margin, then the nasal half is obscured, and, finally, the blurring of the temporal half, resulting in the severe cases in the total obscuration of the optic disk—the condition of papilledema; if a measurable swelling of 2 diopters plus, then the condition of "choked disk."

Those patients having brain injuries with an increased intracranial pressure sufficient to produce a dilatation of the retinal veins and a blurring and haziness of the nasal margins of the optic disks, can still be treated successfully by the expectant palliative treatment; but if the ophthalmoscope reveals a still greater pressure, sufficient to cause an obscuration of the nasal and even the temporal halves of the optic disks—that is a beginning papilledema—then it is always advisable and safer to relieve the increased intracranial pressure as early as possible; whether it is due to cerebral edema or to hemorrhage, the principle remains the same. In these latter patients a decompression and drainage is advisable, not only to save the life of the patient by avoiding a medullary edema, but to lessen the severity and number of the post-traumatic conditions so frequently following a prolonged increase of the intracranial pressure.

Unless the intracranial pressure is very high, resulting from a large, rapid hemorrhage, it is very unusual for the ophthalmoscope to reveal marked changes in the fundus within three hours after the injury. The veins may become full and dilated, but it is rare for an obscuration of the details of the optic disks to occur within this period; if it does, then an immediate decompression and drainage is most advisable. In this series of 487 patients, it was observed that fractures of the occipital bone beneath the tentorium and around the foramen magnum were usually responsible for this rapid and high increase of the intracranial pressure, due possibly to a blockage of the aqueduct of Sylvius, and consequently the ventricle, by either a subtentorial hemorrhage or a cerebellar edema—that is, similar to the signs of pressure resulting from subtentorial lesions blocking the ventricles, whether a tumor, an abscess or a cyst. Frequently the intracranial pressure may become so high that the extracranial vessels in the scalp, and especially of the upper eyelids, become filled and dilated, due to the blockage of the venous circulation intracranially. The prognosis is very poor in these patients, operation or no operation; an early medullary edema is the usual outcome, these patients dying within six to ten hours after the cranial injury.

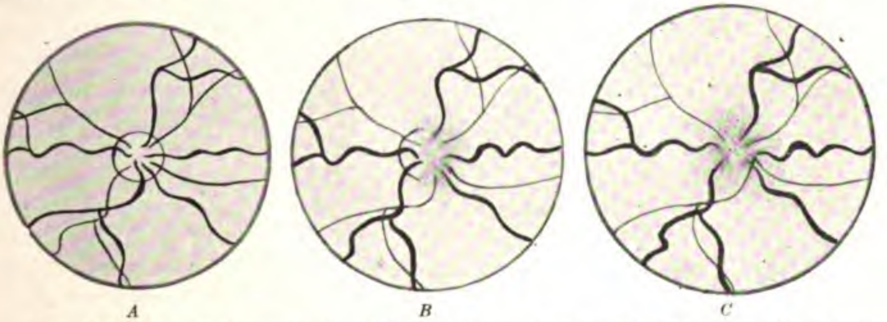

FIG. 14.—A schematic representation of the ophthalmoscopic picture of the fundus of the right eye.
A. Normal fundus—details of optic disk clear, retinal arteries and veins of normal size.
B. Marked increase of the intracranial pressure—nasal half of optic disk obscured by edema, retinal arteries small and retinal veins enlarged.
C. High increase of the intracranial pressure—a papilledema—all the details of the optic disk obscured by edema, and if a measurable swelling above two diopters, then the condition is called "choked disks"; the retinal arteries are small, while the retinal veins are dilated, tortuous and in places buried in edematous tissue. A secondary optic atrophy from new tissue formation results, in varying degree, if this latter condition is permitted to persist for a period of weeks, and surely of months.

It is, however, in those patients who do not show marked signs of high intracranial pressure until three hours or more following the injury that a better prognosis can be given. Repeated ophthalmoscopic examinations are essential—at least once every hour—and if the changes in the fundi advance beyond a fulness of the retinal veins and an edematous blurring of the nasal halves of the optic disks, then an immediate decompression and drainage is advisable. Fortunately, however, with the aid of the expectant palliative treatment the intracranial pressure does not increase beyond this degree in almost 70 per cent. of the patients having brain injuries, with and without a fracture of the skull, so that no operation is necessary in seven out of ten patients, and the recovery will be uneventful with medical treatment alone. It may require four to six days, and even longer, for these signs of a moderate increase of the intracranial pressure to subside, but apparently this duration of moderate pressure does not produce any harmful effects.

Rarely do these fundal examinations reveal a marked increase of the intracranial pressure within six hours after the cranial injury; this is due to the presence of shock in these patients, who later exhibit the marked signs of intracranial pressure. As cranial injuries are usually accompanied by shock of varying degrees, it follows that in these patients the blood-pressure is low, so that even if a large intracranial vessel was torn, yet there could be only a comparatively small amount of hemorrhage, because the resulting increased intracranial pressure would soon be greater than this lowered blood-pressure of shock, and therefore the bleeding would cease. However, as the patient recovers from the condition of extreme shock, then the blood-pressure would rise, and now more bleeding could occur intracranially until the intracranial pressure would again equal the lowered blood-pressure. Finally, if the patient survived this condition of shock, then the blood-pressure would be continuously greater than the intracranial pressure, so that this resulting increased intracranial pressure would produce its characteristic signs in the fundus of the eye—a dilatation of the retinal veins and an edematous blurring of the nasal and temporal margins, then the nasal halves, and, if still higher, then an edematous obscuration of the temporal halves of the optic disks. A measurable papilledema and "choked disks" occur in these patients only when the intracranial pressure is extreme, due to a large intracranial hemorrhage of slow formation, such as the extradural middle meningeal type—just as in brain tumors, or when the ventricles are blocked, producing an internal hydrocephalus. Hence, if the intracranial hemorrhage forms very rapidly and of large amount, the patient usually dies within two or three hours, so that "choked disks" have very little time to be produced. Again, the shock following head injuries usually lasts for about six hours in the patients who survive, and therefore it is rare within these first six hours for the ophthalmoscope to reveal definite signs of an increased intracranial pressure. It may be also noted that the patients who do not survive the condition of shock usually die within the first six hours. It is, therefore, of the greatest importance to recognize these early signs of increased intracranial pressure by repeated ophthalmoscopic examinations, and to realize that these edematous blurrings of the optic disks are more than being merely within physiological limits. It is true that in cases of myopia there is normally an obscuration of the disk outlines, but these patients can be excluded by the measurement of the cerebrospinal fluid by means of the spinal mercurial manometer, which should always be used in these patients.

15. *Lumbar Puncture Findings.*—The normal pressure of the cerebrospinal fluid is 5–9 mm. as registered by the spinal mercurial manometer at lumbar puncture. Any increase over 12 mm. may be considered as being above physiological limits, and therefore indicating a pathological condition within the cerebrospinal canal. The use of the ophthalmoscope is important in these patients in corroborating the lumbar puncture findings, but negative fundi do not exclude the presence of an increased intracranial pressure of mild degree, the ophthalmoscope being a more crude and less accurate means of determining the milder increases of intracranial pressure than the spinal mercurial manometer. It is this estimation of the intracranial pressure which is of importance in the treatment of patients having brain injuries—whether the expectant palliative treatment to facilitate the absorption of mild increases of intracranial pressure by natural means will suffice (and it does in almost 70 per cent. of the patients having brain

injuries), or the necessity for an early mechanical lowering of the high intracranial pressure by means of the cranial operation of subtemporal decompression and drainage (indicated in only about one-third of the patients) in order not only to obtain a larger percentage of recovery of life, but also to secure as normal an individual as before the injury.

The presence of free blood in the cerebrospinal fluid at lumbar puncture in these patients is only of importance as an added sign of the intracranial injury, and merely denotes bleeding from an intradural vessel, with and without a fracture of the skull. The absence, however, of free blood in the cerebrospinal fluid does not exclude an intracranial hemorrhage, and even a subdural and subarachnoid hemorrhage. This observation has been repeatedly made, both at lumbar puncture and then at the later cranial operation. It would seem that in some of these patients there was a blockage of the normal descent of the cerebrospinal fluid into the spinal canal, and thus the intracranial hemorrhage was not demonstrated at lumbar puncture; naturally an extradural hemorrhage of the middle meningeal type does not in itself cause free blood to be revealed in the cerebrospinal fluid. The presence of blood in the cerebrospinal fluid, therefore, is of no importance in the treatment of the patient regarding the advisability or not of a cranial operation, which procedure depends entirely upon the presence of a high intracranial pressure which is not considered absorbable by natural means under the expectant palliative treatment, and the presence or not of blood in the cerebrospinal fluid or of a fracture of the skull (unless it is a depressed fracture of the vault) is of little or no importance in the treatment of these patients. This attitude toward the treatment of brain injuries cannot be too strongly emphasized, for the opinion has become firmly rooted in the medical profession that in the presence of blood in the cerebrospinal fluid, and the definite signs of a fracture of the skull, then the treatment by one extreme group is the expectant palliative one—no cranial operation under any circumstances, even when associated with a high intracranial pressure; whereas the other extreme group would advocate a cranial operation merely because a fracture of the skull and the presence of blood in the cerebrospinal fluid indicated a serious intracranial condition, whether there was an increased pressure or not. These two extreme views should be modified until it is definitely recognized that the degree of intracranial pressure, as ascertained by the ophthalmoscopic and spinal manometric examinations, together with the general condition of the patient, is the chief and deciding factor in the method of treatment of these patients.

The measurement of the pressure at lumbar puncture, therefore, is of the greatest importance, and it should be performed in each patient as early as possible after the signs of initial shock have disappeared. If a lumbar puncture is performed during the stage of initial shock, then the procedure is merely an added shock to the patient, while the pressure of the cerebrospinal fluid in the severe degrees of shock is always normal and even subnormal—as low as 3–5 mm.; the blood-pressure being subnormal from the shock, then the intracranial pressure is also low from the same cause, and no extensive intracranial hemorrhage or cerebral edema can occur until the period of shock has been survived.

The condition of initial shock having disappeared, as indicated by the mperature and blood-pressure ascending to normal and above, and the pulse- and respiration-rates descending to and below 100 and 26 respectively, then it is advisable in these patients to examine repeatedly the fundi ophthalmoscopically, and to measure the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer. A mild increase of the intracranial pressure is usually present, but in only about one-third of the patients will the ophthalmoscope disclose an edematous blurring of the nasal halves of the optic disks, and the spinal manometer register a pressure over 16 mm.—that is, it is only in these 30 per cent. of the patients having brain injuries that the operation of cranial decompression and drainage is advisable, in order to obtain a larger percentage both of recovery of life and of the former good health and normality; whereas the larger number of the patients—about two-thirds of them—do not reveal an increased intracranial pressure or a higher pressure than is disclosed by the ophthalmoscope in the enlargement of the retinal veins and the obscuration of the nasal margins of the optic disks, while the spinal manometer usually registers a pressure approximating the normal, and within physiological limits (10 mm. in adults), or not higher than 12 mm. It is in these latter patients, in whom the pressure does not exceed 16 mm., that the expectant palliative method of treatment is sufficient for an excellent recovery of life and former mentality to be obtained, and it is only in about 30 per cent. of the patients that the intracranial pressure exceeds 16 mm., and these are the ones to whom the operation of subtemporal decompression and drainage offers the highest percentage of recovery of life and the best chance of ultimate normality. In the absence of a marked increase of the intracranial pressure, it is only in those patients having the symptoms and signs of localized cortical irritation, due to a small circumscribed supracortical or cortical hemorrhage and cortical edema sufficient to cause a definite cortical impairment, such as paralysis or convulsive seizures, that the operation of cranial exploration and drainage is advisable. This operative procedure is usually best performed by means of the subtemporal route. Depressed fractures of the vault of the skull producing similar cortical impairment necessitate the early operative removal of the depressed area of bone, and, if associated with a marked increase of the intracranial pressure, then a subtemporal decompression and drainage would be indicated first, to be followed at the same operation by the removal of the bony depression of the vault. It must be remembered that an extensive laceration of the cortex can occur with only a mild increase of the intracranial pressure, due to a small amount of resulting hemorrhage and edema of the adjacent cortex. If the motor area of either cerebral cortex is thus impaired, and particularly the cortex of the left hemisphere, which is possibly more highly developed in right-handed patients than the right cerebral cortex, then the paralysis may be very pronounced, and yet only mild signs of an increased intracranial pressure be revealed. As a rule, however, the signs of an increased intracranial pressure in these patients are very definite, and frequently above 16 mm., owing to the associated free hemorrhage and the extensive cerebral edema of the contiguous cortex and subcortical nerve tissues, and therefore the operation of decompression and drainage is advisable to lower the pressure and thereby obtain the greatest return of cerebral function of the cortical cells, which have not been destroyed but merely compressed by the local hemorrhage and edema—that is, only functionally impaired.

The highest pressure registered by the spinal mercurial manometer in this series of patients having brain injuries was 44 mm. The patient was an unusually able-bodied Italian laborer, of 36 years of age, and only semi-conscious. The ophthalmoscope disclosed the condition of choked disks of 3 diopters. At the operation of subtemporal decompression and drainage upon the seventh day after the injury, a profuse subdural hemorrhage was evacuated, while the cerebral edema was extreme. Fortunately, the patient made an excellent recovery. (*Vide* Case 46, page 256.)

The lumbar puncture when used therapeutically to remove the excess cerebrospinal fluid and free blood in the mild cases of increased intracranial pressure, and thereby improve the general condition of the patient by lessening the severe headache, restlessness, etc., frequently revealed the pressure of the cerebrospinal fluid as being 14 to 16 mm. at the beginning of the puncture drainage; and after 15 to 20 c.c. were removed, then the pressure would be only 10 to 12 mm. This temporary improvement would last, as a rule, only about 12 hours after the first three or four punctures; and then if this means of lessening the pressure, together with the expectant palliative treatment, was sufficient, the registration of the spinal manometer would become lower and lower, until it was no longer necessary to continue the drainage by lumbar puncture. The patients who can be satisfactorily treated by this method are comparatively few, and yet it is a drainage procedure frequently applicable in new-born babies and children, and in the milder conditions of increased intracranial pressure in adults, producing the symptoms and signs of severe headache, extreme restlessness, vertigo, nausea, vomiting, etc. This method should never be substituted, however, for the cranial operation of decompression and drainage in the patients having a high intracranial pressure (over 16 mm.)—the risk of a direct medullary compression in the foramen magnum would be too great.

16. *Traumatic Cerebral Edema.*—The pathology of traumatic cerebral edema is both obscure and puzzling. It may be similar to a localized traumatic edema as occurs elsewhere in the body: a blow upon the arm causes that part of the arm to swell, so that a "bump" is present; this swelling is due to congestion of the neighboring blood-vessels, an outpouring of blood serum from the small capillaries, and is also caused by an increase of the lymph, due possibly to a retarded lymph-flow. In cranial injuries, however, and well illustrated in the more severe forms of so-called "concussion," there occurs frequently a mild edema of the brain—that is, there is a slight increase of the intracranial pressure, as ascertained both by the ophthalmoscopic examinations of the fundi of the eyes, revealing an edematous blurring of the optic disks of varying degree, and by the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer. The latter test reveals in these patients a definite increase in the amount of fluid, bathing, and buoying the central nervous system.

Is this increase of subarachnoid fluid following head injuries merely an increase in the amount of cerebrospinal fluid secreted, or is it a temporary retardation of the excretion of the cerebrospinal fluid through its normal channels—the cortical veins chiefly, the sinuses, Pacchionian bodies, and possibly the lymphatics—just as in the more permanent blockage of cerebrospinal fluid that occurs in the external type of hydrocephalus? Or is this "wet" serous condition of the brain following trauma merely an outpouring of blood serum from the congested and dilated blood-vessels? Or could it possibly be influenced by the presence of a retarded flow of lymph in the brain, or an increase in the amount of lymph itself?

As neither lymph nor lymph-channels within the cerebrospinal nervous system have ever been demonstrated, the possibility of this last factor must necessarily be, at least for the present, of theoretical interest only. An outpouring of blood serum from the dilated intracranial blood-vessels is a possible factor to some extent. My own impression, however, is that cerebral edema frequently results from an increase in the amount of cerebrospinal fluid secreted by the choroid plexus chiefly, and also,—and I believe this is the main cause of traumatic cerebral edema,—from the partial blockage of the excretion of cerebrospinal fluid, due to congestion and a temporary retardation of the flow of intracranial blood in the cortical veins and sinuses. The latter cause in itself is sufficient to produce the condition of a "wet," edematous, water-logged brain of varying degree, and of such frequent occurrence in cranial injuries—that is, traumatic cerebral edema is really an increase in the amount of cerebrospinal fluid both in and around the cerebral tissues.

The presence of an increase in the number of cells in the cerebrospinal fluid in these conditions would merely indicate a certain degree of meningeal irritation, due both to the trauma and to the vascular congestion. At times, a meningismus producing a stiffened neck and even Kernig's sign may be elicited in the more severe conditions of so-called "concussion." It is in these patients with a moderate increase of the pressure of the cerebrospinal fluid that merely a lumbar puncture, and frequently repeated lumbar punctures, suffice to decrease the amount of cerebral edema, so that its symptoms and signs disappear within several days. The question of cerebral edema, however, is not at all settled, and it offers a large field for experimental work.

The condition of chronic cerebral edema persisting for months and even years after the cranial injury is due, I believe, to a partial blockage of the excretion of the normal amount of cerebrospinal fluid into the cortical veins and sinuses, resulting from the organization of subdural and subarachnoid hemorrhage occurring at the time of the cranial injury. In the patients in whom the blockage is sufficient to produce the condition of mild external hydrocephalus, and thus a resulting increase of the intracranial pressure, the pathology as disclosed at operation to lower this pressure is a connective tissue thickening and cloudy induration about the vessel walls —the supracortical veins and sinuses—and especially in the sulci, so that the stomata of exit of the cerebrospinal fluid in their walls are partially blocked and thus the condition of a mild external hydrocephalus develops similar to that type of external hydrocephalus following a mild meningitis when the ventricles are not blocked.

CHAPTER IV

THE SIGNIFICANCE OF INTRACRANIAL PRESSURE

UNDER normal conditions, the height of intracranial pressure depends upon the general arterial blood-pressure in a direct ratio—the higher the blood-pressure the higher the intracranial pressure, and the lower the blood-pressure the lower the intracranial pressure. This ratio remains constant unless marked pathological lesions occur intracranially, such as a large hemorrhage or the terminal stages of a large tumor formation; then the increased intracranial pressure may exceed the general arterial blood-pressure, temporarily and periodically at first, but if not relieved, then permanently, resulting eventually in the death of the patient. Naturally, the intracranial pressure is highest in the arteries, and then in the capillaries, and lowest in the cortical veins and the large venous sinuses. Other conditions remaining the same, any increase in the amount of inflow of blood, or any blockage of its outflow, produces a rise of intracranial pressure.

Another factor, however, in intracranial pressure is the cerebrospinal fluid. Secreted by the choroid plexus of veins in the third ventricle, it passes into the lateral ventricles and also backward through the aqueduct of Sylvius into the fourth ventricle, where it escapes through the foramina of Majendie and Luschka into the subarachnoid spaces to bathe, as it were, the cortex of the brain and the surfaces of the spinal cord—floating them to a certain extent. It is now believed that the cerebrospinal fluid re-enters the circulation chiefly by means of the cortical veins and also through the Pacchionian bodies situated along the longitudinal sinus. Whether this fluid brings nourishment to the nerve cells or carries away waste products is unknown. The amount of cerebrospinal fluid depends upon many factors, especially the rate of its secretion and the rate of its excretion. In the most common types of external hydrocephalus, the increased amount of cerebrospinal fluid is due to a lessened excretion by blockage of its stomata of exit in the cortical veins and sinuses. Temporarily, at least, the amount of cerebrospinal fluid tends to be increased by a rise in the blood-pressure; especially is this so in traumatic conditions of the skull and particularly in brain injuries with and without a fracture of the skull.

One effect of a prolonged increase of the intracranial pressure is the resulting partial anemia of the cortex; the amount of blood reaching the cortex is thus lessened, so that the delicate cortical nerve cells do not receive their normal blood supply; a partial "starvation" of them results, so that the cortex becomes pale, and if this condition persists for any length of time, then an increase of neuroglia cells occurs in it. This increase of neural connective tissue tends to prevent the normal functioning of the cortical nerve cells, and is, in my opinion, the cause of many of the so-called "post-traumatic neuroses" following brain injuries, such as persistent headaches, dizzy spells, indefinite pains in the head, changed personality to one of emotional excitement and irritability or to one of depression, general nervous instability, fainting spells and even epilepsy itself. A high intracranial pressure persisting ten days or more is apparently sufficient to produce these conditions. One case at autopsy, following a brain injury with fracture of the skull 8 years previously, showed a marked increase of the neuroglial tissue cells in the cortex; since the cranial injury, beside the definite signs of an increased intracranial pressure, the patient had suffered from intense headache and at times even epileptiform attacks. There may have been in this patient subpial punctate hemorrhages causing an unusual amount of connective-tissue formation in the cortex.

A number of years ago, Kocher, by careful observations, recognized the symptoms and signs of increased intracranial pressure and divided clinically the resulting intracranial compression into four main stages. These are:

The First Stage of Compression, Being the Medical Stage of Compensation.—The effects of increased intracranial pressure vary according to whether the compression is sudden and acute, or gradual and chronic; if the latter, then a certain amount of adaptation of the brain occurs, so that a much higher pressure may be endured without producing the marked signs of its presence; this occurs very frequently in slowly growing tumor formations of the brain. In either case, whether acute or chronic, the increased intracranial pressure first expels the excess cerebrospinal fluid and, as brain tissue itself is non-compressible, it then compresses the local blood-vessels, so that the amount of intracranial blood is slightly lessened. As the blood in the cerebral veins is under a very low pressure, these veins become filled with blood and dilated, so that the next sign is a venous stasis—its symptoms being headache, drowsiness, and possibly stupor; the pulse, respiration and blood-pressure are not affected; the retinal veins become dilated. (Even in this first stage, a slight haziness and edematous blurring of the nasal margins of the optic disks may be observed. The pressure of the cerebrospinal fluid by the spinal mercurial manometer may register 12–14 mm. of mercury. Naturally, the expectant palliative treatment is indicated, and in over two-thirds of the patients this treatment alone suffices to insure an excellent recovery in this *medical* stage of increased intracranial pressure.)

The Second Stage of Compression, Being the Ideal Operative Stage.—If, however, the intracranial pressure still rises, it tends to approximate the pressure in the capillaries, and so a partial anemia results. If the pressure is a local one, such as that due to a middle meningeal hemorrhage or a depressed fracture of the vault, then a local anemia of the underlying cortex results with impairment of function of that cerebral area. Naturally, the more distant the areas of the brain are from the localized compression the less are they affected, and as the falx cerebri and the tentorium form three fairly separate compartments of the brain, it is possible for one hemisphere to be disabled by an extradural hemorrhage, and yet the opposite hemisphere and especially the cerebellum and medulla situated beneath the tentorium to be only slightly affected; the tentorium is of the utmost importance in this respect—a protecting barrier for the all-important medulla. If, on the other hand, the increased intracranial pressure is of subdural origin, due to a subdural hemorrhage or a sudden increase in the amount of cerebrospinal fluid following a brain injury with and without a fracture of the skull, then the pressure becomes general and all portions of the brain are equally affected. In the case of a subdural clot, naturally the underlying cortex is more compressed than the more distant areas of the brain, although even in these cases of general pressure it is the effect upon the medulla that is to be feared; subtentorial local pressure of moderate severity produces the same medullary impairment as high intracranial and supratentorial general pressure.

The first effect upon the medulla of a continued rise of the intracranial pressure is one of slight anemia of the medulla producing a slow pulse of 60 or lower due to the stimulation of the vagus nucleus, and a slight rise in the general arterial blood-pressure due to the stimulation of the vasomotor centre, causing not only a constriction of the peripheral blood-vessels themselves, but especially of those vessels of the splanchnic field. In addition, the intracranial venous stasis becomes more marked so that the headache becomes severe and associated with restlessness and even delirium; a definite cyanosis appears; an ophthalmoscopic examination reveals large dilated retinal veins with or without edema of the optic disks. These definite, though moderate, signs of high intracranial pressure form the second stage of compression clinically, which is undoubtedly the best time to operate to relieve the intracranial pressure—before the extreme signs of medullary compression have occurred. (In this operative stage of intracranial pressure, the more modern methods of examining the fundus of the eye with the electrical ophthalmoscope (the direct method) rarely fails to disclose a definite edematous obscuration of the optic disk outlines—a mild papilledema; the spinal mercurial manometer usually registers an increased intracranial pressure above 15 mm. of mercury up to 22 mm., and at times even higher. Marked stupor usually appears; unconsciousness may or may not be present; if it is present, then it is rarely continuous, but is more frequently of the periodic type—an extreme stupor from which the patient can be roused and then again cannot be roused by the usual methods of supraorbital pressure, inhalation of ammonia, etc. This second stage of compression is the *ideal* operative period both as to immediate recovery of life and the permanent recovery of unimpaired function; it anticipates the dangerous stages of extreme medullary compression and of medullary edema and collapse.)

The Third Stage of Compression, Being the Imperative Operative Stage of Medullary Compression.—The third stage of compression clinically consists of the major or bulbar (medullary) signs of compression. As the intracranial pressure continues to rise, it produces a greater anemia of the medulla so that the intracranial pressure at times may equal the capillary pressure of the medulla; if it were not for the regulatory mechanism of the circulation in the medulla, such an occurrence would result in the immediate and permanent cessation of the cardiac and pulmonary activity, and, therefore, the death of a patient. Fortunately, however, as the anemia of the medulla becomes greater, this very absence of blood so stimulates its vasomotor centre that the general arterial blood-pressure is raised, more blood is forced into the medulla, and in this manner the partial anemia is overcome, at least temporarily. Clinically, the picture is most striking; as the intracranial pressure increases until it tends to prevent the normal flow of blood into the medulla, the resulting partial anemia so stimulates the vagus centre that the pulse-rate gradually becomes lowered to 50 and below and of a full bounding character; the respiration becomes less and less frequent until a period of temporary apnea or non-breathing results from the anemia of the respiratory centre in the medulla: the patient may not breathe for 40 seconds and even longer. During the earlier part of this period of "down-wave" of the pulse- and respiration-rates, the blood-pressure falls slightly, the patient gradually becoming more and more stuporous, the pupils slowly dilating, and the reflexes being abolished—the results of a definite anemia of the medulla. Then, as this prolongation of the medullary anemia stimulates its vasomotor centre to renewed activity, the general blood-pressure is gradually raised to overcome the intracranial pressure until blood is forced into the medulla, the heart-rate increases, and then the patient begins to breathe again as a result of the respiratory centre being resupplied with blood. During this period of "up-wave," the cyanosis is extreme, the pupils contract, and the patient may groan, become restless and even regain consciousness; the reflexes return and the ophthalmoscopic examination reveals a double papilledema or "choked disks"; that is, the intracranial pressure becomes so high that the resulting venous stasis produces an edema of the optic disks so that their margins and the entire disks themselves are obscured and the retinal veins are dilated and at times buried in the edematous retina; even the other extracranial veins of the scalp, and particularly of the upper eyelids, are dilated. As the medulla becomes supplied with blood again following this period of "up-wave" of the pulse-, respiration- and blood-pressure-rates, the stimulation of its vasomotor centre is lessened so that gradually the general arterial blood-pressure diminishes until the symptoms and signs of the "down-wave" become more and more marked; then the "up-wave" begins again, as outlined above, and this periodicity of symptoms and signs depending upon the rise and fall of the general blood-pressure causing the Cheyne-Stokes type of pulse and respiration (Traube-Herring waves) occurs again and again. This condition may continue for hours. (This third stage of intracranial compression may be known as the period of *imperative* operation: unless an immediate decompression and drainage is performed, it is most rare for these patients to survive; even with operation, the chances for recovery are doubtful. The ophthalmoscope reveals a measurable papilledema—even above 2 diopters frequently—that is, to a degree of "choked disks." The spinal mercurial manometer registers a pressure above 20 mm. of mercury and frequently even 30 mm. Unconsciousness is almost always present, either periodic or continuous, especially during the period of "down-wave," and always during the later and advanced period of this stage. This stage of extreme intracranial pressure should be anticipated if possible and it usually can be, by the more modern methods of examination and estimation of the intracranial pressure as outlined above; no patient should be permitted to enter this stage of compression if it can be possibly avoided—the mortality being very high.)

Fourth Stage of Compression, Being the Non-Operative Stage of [illegible] *lary Edema.*—Unless this high intracranial pressure is quickly relieved by an operation (and even with an operation the chances for recovery at this stage of compression are slight), this regulatory mechanism of the medulla will finally become fatigued, so that the vasomotor stimulation will no longer be able to raise the general arterial blood-pressure above the intracranial pressure, and thus during one of the "down-waves," a permanent fall of blood-pressure will occur; respiration will no longer begin again, and the heart will continue to beat irregularly and rapidly as a separate organ until the blood-pressure gradually falls to zero, so that even the heart itself will cease beating. This stage of respiratory paralysis, associated with rapid and irregular cardiac efforts, dilated pupils, profound coma and complete muscular relaxation and a permanent fall of the general arterial pressure, forms the fourth stage clinically of a permanent anemia of the medulla—the stage of loss of compensation or the terminal stage, always resulting in the death of the patient. (This fourth and last stage of intracranial compression might be well known as the *non-operative* or *moribund* period. Once these patients have entered this stage of medullary edema as shown by a rapidly increasing and irregular pulse-rate above 120, a low blood-pressure descending to 100 and even lower, and by a rapidly increasing, irregular and shallow respiration, then these patients all die—operation or no operation; in fact, any operation in this period merely hastens the exitus and so tends to discredit cranial surgery. A pulmonary edema is usually considered the immediate cause of death, although it is really secondary to the medullary edema and collapse. In this series of patients having brain injuries with and without a fracture of the skull in whom the fourth stage of medullary edema was observed (and these patients all died whether they had been operated upon or not), it was exceedingly rare for a permanent respiratory paralysis to occur during a period of apnea to the extent that death occurred at that time; the usual observation was: a lowered and irregular pulse- and respiration-rate of the Cheyne-Stokes type continued until the pulse-rate began to ascend rapidly to 120 and above, and at the same time associated with an increasing respiration-rate to 40 and higher—this sudden change and onset of medullary edema occurring within a period of several hours; the pulse- and respiration-rates continued to such a height and irregularity that soon the pulse could not be palpated and even the heart-beat became imperceptible, whereas the respiration now faded into a shallowness that it was not possible to auscultate; a pulmonary edema—choking and drowning the patient, as it were, in the body fluids—most frequently occurred in heavy obese patients, and especially in alcoholics.)

CHAPTER V

THE SIGNS OF INTRACRANIAL PRESSURE OBSERVABLE IN THE FUNDUS WITH THE OPHTHALMOSCOPE

THE fundus of the eye, and particularly the retina, being an offshoot of the brain, is most intimately connected with the brain and the intradural cavity, so that any lesion within the intracranial cavity which increases its normal content would naturally tend to be shown in the fundus of the eye, especially about the optic nerve head; that is, unless the normally free communication within the vaginal sheath between the intradural cavity and the optic papilla is obstructed by local disease, adhesions, etc., we should expect the signs of increased intracranial pressure to be observable in the fundus of the eye, particularly about the optic disk. Again, an increased intracranial pressure sufficient to retard and even prevent the normal return flow of blood in the retinal veins would also tend to cause a dilatation of these retinal veins and the usual condition resulting from their dilatation and congestion.

The effect of an increase of intracranial pressure upon the fundus of the eye can be very easily demonstrated in its various stages by the experimental production of an internal hydrocephalus in dogs. Three years ago, the condition of hydrocephalus was successfully produced by me in nine puppies of the age of ten days to two weeks; by means of a suboccipital exposure, a small gelatine capsule filled with cotton was inserted into the aqueduct of Sylvius so that the cerebrospinal fluid could not escape from the third and lateral ventricles; a resulting internal hydrocephalus with dilatation of the ventricles, and therefore an increased intracranial pressure, occurred, so that it was possible with the ophthalmoscope to observe the changes in the fundus of the eye due to this increase of intracranial pressure. Within five or six hours after the insertion of the cotton plug into the aqueduct of Sylvius, in each one of the puppies the retinal veins gradually became dilated; apparently the veins over the nasal half enlarged earlier and possibly more than the veins over the temporal half of the retinal fundus. This congestion and dilatation of the retinal veins was the first sign indicative of an increase in the intracranial pressure. Within two to four hours later, the nasal margin of the optic disk would become blurred, then the temporal margin, then the nasal half and finally the temporal half of the disk would become obscured, so that within a period of twelve to twenty-eight hours following the production of a definite increase of intracranial pressure, the details of the optic disks could no longer be observed; in four dogs the edema of the optic disks was so great within this period of time that a measurable swelling could be observed with the ophthalmoscope and thus the designation of "choked disks" could be applied; in the other five dogs, the measurable papilledema occurred from twelve to twenty hours later. In one dog this condition of internal hydrocephalus with high intracranial pressure was not relieved by a subsequent drainage operation, and it is interesting to note that definite signs of a secondary optic atrophy began to appear nine weeks later; the remaining eight dogs were all drained through a subtemporal operation with six linen strands being inserted into the ventricle in order to relieve the condition of internal hydrocephalus; in all but three of the dogs the increased intracranial pressure was relieved, and it was most interesting to note that the subsidence of the choked disks was in just the reverse order of their occurrence—that is, the measurable swelling of the papilledema first disappeared, then the blurring of the temporal half and then of the nasal half of the optic disk; then the temporal margin and later the nasal margin appeared—though slightly blurred in all of the puppies; that is, the persistent dilatation of the retinal veins and some blurring of the nasal margins of the optic disks indicated that the intracranial pressure had not been entirely relieved by the operation. In the remaining three puppies the optic disks remained entirely obscured—only the measurable swelling disappeared, so that in these dogs the operation of drainage to relieve the condition of internal hydrocephalus was not successful.

This experimental work has been most instructive regarding the mechanical factor in the production of papilledema and the condition known as "choked disks." There exists at present much confusion in the terminology of retinal conditions, especially in their relation to intracranial pressure; the terms papillitis and papilledema have been used interchangeably, and "choked disk" most freely and carelessly. Naturally, the terms papillitis, retinitis, and optic neuritis imply a condition of inflammation of the nerve head and the retina. Inflammation (in its modern conception) is due to toxic and infective causes alone, so that in the condition of nephritis, diabetes, and the various forms of meningitis, the term papillitis would indicate a retinal change due to some toxic or infective cause, whereas the blurring and edema of the details of the optic disk due to an increase of intracranial pressure in purely mechanical conditions, such as an intracranial tumor mass and hemorrhage, could be termed as a papilledema; and if a measurable edema, then a swelling of the disk up to the stage of "choked disk." In order that a choked disk occur, it must always be preceded by a series of blurring of the details of the optic disk, so that these early edematous blurrings of the optic disks have been termed the mild or early stages of papilledema or even a "choking" of the disks. If it is believed that intracranial pressure alone does cause in the fundus of the eye an inflammation (using the term "inflammation" in its modern sense as being due to a toxic or infective cause), then the term papillitis is perfectly proper, although the etiological factor in its production is pressure; but I do feel that the modern methods of examination of the cerebrospinal fluid, and especially its cell count, whereby any inflammation and infectious condition within the cerebrospinal canal and also its projecting connections, such as the optic vaginal sheath, can easily be demonstrated,—that these methods should disclose the infective character of the cerebrospinal fluid if that is a cause of the so-called papillitis in the purely pressure cases. It has been frequently demonstrated in the body tissues and elsewhere that pressure can and does cause congestion and edema, but not inflammation (in its modern sense), unless infection is present; pressure upon tissue renders that tissue more susceptible to inflammation by infection—a predisposing cause—but pressure in itself does not mean an inflammation. It is rather rare for retinal hemorrhages to occur in cases of "choked disks" due to even extreme intracranial pressure alone, whereas in conditions of neuro-retinitis, optic neuritis, and papillitis due to toxic causes such as diabetes and nephritis—these hemorrhages occur very frequently. However, if we must take it for granted that the retinal and optic disk changes in conditions of intracranial pressure are due to both pressure and toxic factors—possibly the pressure producing the toxic appearance of inflammation—then the condition of blurring and edema of the details of the optic disks would precede the stage of papillitis; and if the papilledema became measurable to two or more diopters, then the term "choked disks" should be applied. In discussing the pressure signs observable in the fundus in the following intracranial conditions, I shall use the terms blurring and edema of the details of the optic disks to indicate the earlier stages of increased intracranial pressure, and its later stages of measurable swelling of the optic disks by papilledema, and the term "choked disks" in the conditions of extreme intracranial pressure where the papilledema is greater than two or three diopters. These signs of increased intracranial pressure as exhibited upon the fundus of the eye have been checked up and confirmed by a measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of a spinal mercurial manometer, so that when there is observed a blurring or edema of the margin of the nasal half of the optic disk in a patient following a cranial injury, or in a patient in whom a brain tumor or brain abscess is feared, or in a baby following a difficult labor and convulsions occur so that an intracranial hemorrhage is suspected, then it is very important to record accurately the pressure of the cerebrospinal fluid at lumbar puncture; if the pressure of the cerebrospinal fluid, too, is shown to be increased and thus the ophthalmoscopic findings are confirmed, we can then reach a more accurate diagnosis of the intracranial condition and advise accordingly. Naturally, in normal fundi, blurring and mild obscuration of the details of the optic disks occur and are considered as being within physiological limits; especially is this true in myopia, but if this obscuration of the details of the optic disks is observed and then the measurement of the cerebrospinal fluid is performed at lumbar puncture by means of a spinal mercurial manometer (the most accurate method now known to record the pressure of the cerebrospinal fluid), we are thus enabled to exclude those cases of so-called normal blurring of the optic disks.

There are several intracranial conditions that frequently produce definite pressure signs observable in the fundus of the eye. In order to understand thoroughly and to appreciate the significance of the various degrees of intracranial pressure as revealed in the fundi, the ophthalmoscopic findings of the following intracranial conditions are discussed briefly and in this order: brain tumor, brain abscess, hydrocephalus, selected cases of cerebral spastic paralysis due to an intracranial hemorrhage at the time of birth, and lastly an intracranial hemorrhage and cerebral edema following brain injuries with and without fracture of the skull.

I. *Brain Tumor.*—The condition of "choked disk," and, if not relieved, its subsequent secondary optic atrophy, is well known in patients having the signs of intracranial tumor. Naturally, for a "choked disk" to occur, there must be a very high intracranial pressure, and I believe it is rare in cases of brain tumor for a "choked disk" to result unless the tumor has become of very large size, or it causes a blockage of the ventricles, and thus produces an internal hydrocephalus, such as the posterior mid-brain tumors and the subtentorial tumors and cysts. It is comparatively easy to make the diagnosis of brain tumor at this stage of papilledema, and I feel that if these patients had been examined ophthalmoscopically early, then the more mild pressure signs observable in the fundus resulting from the smaller tumor mass would have been ascertained and the patient thus spared an impaired vision, if not blindness itself. The stage of "choked disk" must naturally be preceded by the earlier and milder stages of disk blurring and papilledema, and should therefore be recognized as being more significant than being within physiological limits. Only too frequently the surgically successful removal of the brain tumor is possible, and yet the patient has already been irreparably damaged by the non-recognition of its pressure signs until it is too late for a normal person to be obtained. An interesting syndrome ophthalmoscopically is that of certain frontal tumors, which may in their growth by direct pressure down upon the ipsolateral optic nerve produce a *primary* optic atrophy, and as the result of the increased intracranial pressure there is observed in the opposite fundus a "choked disk"—the forerunner of a *secondary* optic atrophy.

II. In *brain abscess*, there is a replacement and substitution of cerebral tissue by the purulent detritus, and thus, as in gliomatous tumors which infiltrate and replace brain tissue rather than push it aside, it is rare for the definite signs of intracranial pressure to be observed in these patients unless, as has been stated before, the ventricles are blocked, or a toxic and infective meningitis occurs from the presence of the abscess. This ventricular blockage is of frequent occurrence in subtentorial and cerebellar abscess, but it is rare for an abscess of the temporo-sphenoidal lobe, which is the most common location of brain abscess following the usual cause—an otitis media—to produce fundal changes even though the abscess may reach the size of an orange and even larger.[1]

III. *Hydrocephalus.*—The ophthalmoscopic findings in the cases of hydrocephalus—whether it is of the internal type due to a blockage of the cerebrospinal fluid in the ventricles and thus producing ventricular dilatation and its resulting extreme intracranial pressure, or of the external type of hydrocephalus which is due to a partial blockage of the escape of the cerebrospinal fluid from the general cerebrospinal canal. This latter condition rarely causes a measurable papilledema, whereas the internal type of hydrocephalus can produce "choked disks" of extreme degree and their subsequent secondary optic atrophy. Fortunately in little babies before the sutures have firmly united, the skull itself can enlarge, and thus in many of these patients a natural compensatory "decompression" takes place so that the peripheral vision may not be impaired; a drainage operation, however, offers these children their best chance of approximating normality.[2]

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[1] *The Laryngoscope*, March, 1914.

IV. Selected cases of *cerebral spastic paralysis* due to an intracranial hemorrhage at birth.—In these patients, the ophthalmoscopic examination is of very great importance in aiding the differentiation of this type of intracranial lesion from the other causes of cerebral spastic paralysis. It is very interesting to note that Mr. W. J. Little, in his first monograph in 1843 upon spastic paralysis, entitled "Deformities of the Human Frame,"[3] or the now so-called Little's disease, stated that the condition was due to an impairment of nerve tissues resulting from their lack of development and also to an earlier meningitis; a few cases, however, followed difficult labors, and undoubtedly these were, in his opinion, due to an intracranial hemorrhage at birth. In 1862 (nineteen years later), in his second monograph upon spastic paralysis, entitled "On the Influence of Abnormal Parturition, Difficult Labors, etc., upon the Mental and Physical Condition of the Child,"[4] he says that, in his opinion, almost 75 per cent. of these cases are due to intracranial hemorrhage. Recent investigation of this condition also confirms this belief that about three-fourths of these cases are due to intracranial hemorrhage at the time of birth.

In order to differentiate the three chief causes of cerebral spastic paralysis in children—that is, first, a lack of development of the cerebral cortex or its pyramidal tracts; secondly, a meningitis and meningo-encephalitis following the infectious diseases such as cerebrospinal meningitis, measles, scarlet fever and whooping-cough; and lastly, an intracranial hemorrhage, it is very important that a careful ophthalmoscopic examination should be made as early as possible. The measurement of the pressure of the cerebrospinal fluid at lumbar puncture with the spinal mercurial manometer should also be used to confirm the fundal findings of increased intracranial pressure. Naturally, in cases of lack of development of cerebral tissues there can be no increase of the intracranial pressure, and this is also true of those patients who have survived a meningo-encephalitis—a destruction and atrophy of cortical nerve tissue. On the contrary, if an intracranial hemorrhage has occurred, then there should be signs of an increased intracranial pressure as a result of the hemorrhage, as shown by a dilatation of the retinal veins and an edematous blurring of varying degree of the optic disks. Naturally, the earlier this examination is made the more definite are the fundal signs of intracranial pressure, whereas in the older children the ophthalmoscopic examination may reveal only a dilatation of the retinal veins with thickened walls from new tissue formation and a shallow disk cup; the disk itself is blurred in its details, particularly along the nasal margin, while, the temporal margin, and even the nasal half of the disk, may be obscured. In no patient of over one year of age have I found a measurable swelling of the disk to the degree of "choked disk." Upon examining a patient having cerebral spastic paralysis, if the above fundal changes are noted, then the pressure of the cerebrospinal fluid should be measured at lumbar puncture in order to ascertain whether the fundal changes are due to a local condition within the orbit or are possibly within the normal physiological limits. In these children within one week after birth, there is usually blood in the cerebrospinal fluid at lumbar puncture, as was demonstrated in 21 children upon whom I operated within three days after birth.

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[2] *American Journal of Medical Science*, April, 1917.

[3] *The Lancet*, vol. i, p. 350, December 16, 1843.

[4] *Obstetrical Transactions*, vol. iii, p. 293, 1862.

In a report[5] of 954 cases of cerebral spastic paralysis up to April 1, 1916, only 26 per cent. of them—that is, only one out of every four patients examined—showed these definite signs of an increased intracranial pressure, and the spastic condition was therefore due to a hemorrhage, and in these selected patients by a cranial operation to relieve this increased intracranial pressure upon the brain, an improvement was to be obtained. At that time, I had operated upon 219 children with a mortality of 16—that is, 8 per cent. The history of these children is very suggestive: of the 219 operated patients, only 26 were not first children; only 8 were not full-term babies; only 21 were not born after a difficult labor, with or without instruments; only 49 did not have convulsive twitchings immediately after birth; and in only 21 children was the spasticity noticed before the eighth month after birth. A permission for autopsy is obtained before operation in each patient, both private and ward, and it is by this valuable means, as well as by the operative findings, that the diagnosis is verified and other data ascertained.

V. *Intracranial hemorrhage* and *cerebral edema* following brain injuries, with and without a fracture of the skull, rarely produce a measurable papilledema to the extent of "choked disks." The reason for this is obvious: unlike brain tumor, hydrocephalus, and the other intracranial conditions which enlarge slowly and thus permit the brain and particularly the medulla to adapt themselves to this increased pressure with little immediate risk, in many cases of traumatic intracranial hemorrhage and cerebral edema, in the absence of marked shock, the intracranial pressure rises most rapidly, so that the compensatory mechanism of the medulla has little time to adjust its vasomotor and respiratory centres to this increased pressure, and the result in these patients is death even before the development of "choked disks" is possible. If these patients could survive this greatly increased and rapidly produced intracranial pressure, then a measurable papilledema and "choked disks" would occur. In brain injuries with and without a fracture of the skull, "choked disks" do occur in the cases of large hemorrhage following a rupture of the middle meningeal artery. In these cases of intracranial pressure of comparatively slow production, the medulla can adapt itself to the pressure, and thus death does not occur before a measurable papilledema and "choked disks" are possible. I have operated upon a number of these patients; the "choked disks" of two or more diopters developed within several days after the brain injury; at the operation of subtemporal decompression, either an extradural hemorrhage, alone or associated with a subdural hemorrhage of large amount, was removed. The subsidence of the measurable papilledema began immediately after the operative drainage of the intracranial pressure, so that within 24 hours there were present only an edema and blurring of all the details of the optic disks but not a measurable swelling of the disks themselves.

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[5] *New York State Journal of Medicine*, October, 1916.

It is most rare in these cases of brain injuries with and without a fracture of the skull for an edema of the optic disks to appear within six hours following the trauma, and particularly is this true of those patients in the various degrees of shock; the greater the shock the less the general blood-pressure, and naturally, even though a large intracranial sinus or vessel was torn, yet it would be difficult for any extensive hemorrhage to occur. Just as soon as the intracranial pressure equalled this lowered blood-pressure of shock, then no more bleeding could occur because the intracranial pressure would now be equal and even higher than the blood-pressure; as the patient rallied from the condition of shock, then naturally the blood-pressure would become higher and then more intracranial bleeding would occur, and thus the signs of intracranial pressure, such as edema of the optic disk outlines, would now be possible. In these patients, the marked signs of shock usually last about four to six hours; and again, those patients who cannot survive the condition of shock—they die within six hours after the injury.

In these cases of brain injury it is of little or no importance in the treatment to know the site and extent of the fracture, whether it is a vault or basal fracture, but it is of the utmost importance to ascertain the presence or not of an increased intracranial pressure, both by careful and repeated ophthalmoscopic examinations, and also by the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer. If there are definite signs of a marked increase of the intracranial pressure, such as an edematous blurring of the details of the optic disks and twice the normal pressure of the cerebrospinal fluid and even more, whether the medullary compression signs of a lowered pulse-rate and Cheyne-Stokes respiration are present or not, then I believe an early relief of this increased intracranial pressure by means of a simple subtemporal decompression and drainage is advisable before the patient reaches the dangerous stage of extreme medullary compression and even edema, and thus the collapse of the medulla itself. If an operation is postponed in these patients until a "choked disk" occurs, then the ideal time for operation will have been lost, and it is then very doubtful whether the patient will recover; besides, should the patient having had high intracranial pressure recover without an operation or at best a very late operation, then the danger of post-traumatic conditions is very great indeed. These conditions, due to a prolonged increase of the intracranial pressure, are persistent headache, an emotional instability of either the excited or the depressed type, mental and physical lassitude and early fatigue, and in rare cases even epilepsy in its various forms. It is evident, therefore, in patients having cranial injuries, that it is most important to establish the presence or absence of an increased intracranial pressure—it matters not whether that pressure is or is not due to hemorrhage or cerebral edema—and that repeated ophthalmoscopic examinations are of the greatest aid in facilitating an accurate diagnosis and the early treatment of the condition. Besides the conditions already mentioned, there are still other intracranial lesions in which an ophthalmoscopic examination is of the greatest importance: in cases of the various forms of meningitis, as an aid in differentiating the types of apoplexy, and a most important function in so many conditions—the presence of a negative fundus.

In view of these considerations, therefore, it is essential that careful and repeated ophthalmoscopic examinations of the fundi be made, as they are of the greatest importance in the differentiation of many intracranial lesions; that the signs of moderate intracranial pressure should be recognized, and that it should be realized that "choked disks" occur only as an advanced result of high and prolonged intracranial pressure, and rarely in patients having brain injuries; that the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer is the most accurate means of determining the intradural pressure; and lastly, the intelligent use of the ophthalmoscope, especially the direct method, should be much more intensively studied in the medical schools and in the hospitals than it is at present.

CHAPTER VI

INTRACRANIAL PRESSURE AS MEASURED BY THE SPINAL MERCURIAL MANOMETER AT LUMBAR PUNCTURE

UNTIL three years ago the ophthalmoscopic examination was considered the most satisfactory method of determining the presence or not of, and the degree of, an increased intracranial pressure; during the past three years the significance of the pressure of the cerebrospinal fluid and its accurate registration by means of the spinal mercurial manometer have been so developed that the ophthalmoscopic findings, unless the increased intracranial pressure is of sufficient degree to produce the condition of "choked disks" (a rare observation in these patients having brain injuries), are merely indicative of intracranial pressure, whereas the findings of the spinal mercurial manometer are not only confirmatory, but the degree of increased intracranial pressure can be most accurately established. The most practical of these instruments is the one devised by Landon; it is similar to a blood-pressure apparatus (Fig. 15) and there is no risk to the patient other than that of a lumbar puncture which is practically nil when properly performed. It is essential for an accurate measurement of the pressure that the patient should be lying quietly upon his side with the median line of the head upon an exact level with the spinal canal. (If the physician is right-handed, then it facilitates the insertion of the puncture needle if the patient lies upon the left side.) The thighs and knees should be flexed upon the chest with the head and neck bent forward so that the knees almost touch the head; an assistant facing the patient and putting his right arm about the patient's neck and his left arm under both knees can easily approximate these parts and at the same time hold the back of the patient at the edge of the bed and at right angles to it; in the manner of forcibly "arching" the lumbar region, the spinous processes are diverged, the intervertebral spaces widened and thus the insertion of the puncture needle is not a difficult one; either the mid-line may be used so that the needle is thrust directly at right-angles into the spinal canal, or if it is desired to avoid the interspinous ligaments, then the needle may be inserted just below and lateral to the mid-line (1 cm.) and then slightly obliquely upward until it enters the spinal canal. (In very muscular adults the latter method of insertion may be advisable, but it is not necessary and particularly in children; I have never seen any ill-effects from a lumbar puncture in the mid-line, and it really makes little difference which route is used; at times owing to abnormal bony conformation or in patients whose backs cannot be satisfactorily "arched," then the performance of a successful puncture may be most difficult; either route may be used, and a number of attempts may be necessary before the spinal canal is entered; it is exceedingly rare, however, for a lumbar puncture not to be possible in these traumatic patients.)

A general anesthetic is never necessary, and it is only in the occasional patient that even local anesthesia is advisable—the effect being more of a psychic one than a lessening of the skin prick, which is, in reality, little more painful in this area of the back than the pulling of a hair or a pinch of the skin; if, however, a local anesthetic must be utilized, then the freezing application of ethyl-chloride can be used or a weak novocaine solution—and, of almost equal value for these patients, merely sterile water hypodermically; in babies and children, any procedure of local anesthesia, and especially hypodermically, is more terrifying to the patient than the puncture itself, so that local anesthesia for lumbar puncture is never willingly advised.

The site of election for the lumbar puncture is the fourth lumbar interspace—almost on a level with the crest of the iliac bones; at times the vertebral interspace between the third and fourth spinous processes is a more satisfactory approach to the spinal canal, but a lumbar puncture should not be made between the second and third lumbar vertebræ, for fear of injuring the terminal portion and filaments of the spinal cord, which may be abnormally low; in children and babies, especially, the puncture should be limited to the fourth lumbar interspace between the fourth and fifth spinous processes as the end of the spinal cord may be as low as the fourth lumbar vertebra before it makes its normal ascent as the child grows older to become stationary at about the middle of the second vertebra.

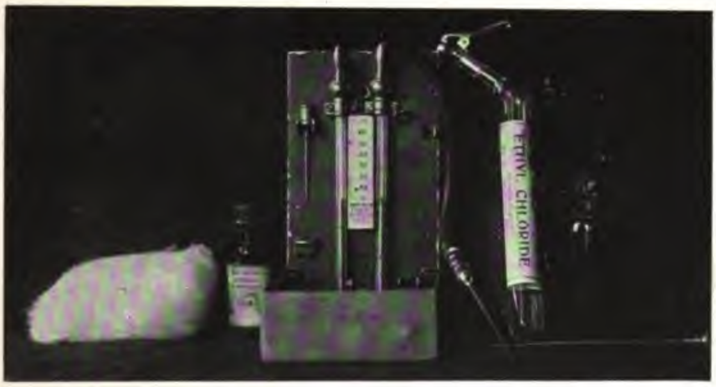

FIG. 15.—The Spinal Mercurial Manometer, consisting of a U-shaped tube containing mercury to the level of zero on the registered scale of cubic millimetres; the lumbar puncture needle, after the withdrawal of its stylet, has been connected with the rubber tubing of one arm of the mercurial tube. The necessary equipment of a small piece of sterile cotton or gauze and tincture of iodine for the asepsis, and whenever advisable for local anesthesia the solution of ethyl chloride or a weak novocaine mixture.

During the past three years, the following technic has been used in over eleven hundred lumbar punctures; the patient lying upon his left side and being "doubled up" by an assistant, so that the back was "arched" by the knees being approximated to the head, a small area of the spine, about one silver dollar in size, is painted with iodine solution, between the fourth and fifth spinous processes—roughly on a level with the iliac crests (Fig. 16). The operator's right thumb and forefinger may be also painted with iodine to minimize the risk of infection in handling the puncture needle, and yet this is not necessary, as the point and shaft of the needle should never be touched, and it is thus possible to insert the puncture needle without touching any portion of it other than the hilt. (This care in not touching the point and shaft of the puncture needle is a much safer precaution than the use of sterile gloves for hands which are not surgically cleansed; the operator may thus consider himself "clean," the puncture needle be handled carelessly and the possibility of an infection greatly increased. Sterile gloves in themselves do not render the hands sterile, and if they are used, then the hands should be scrubbed with green soap and water and made surgically clean in the routine manner.) It is usually possible to know when the puncture needle reaches and passes through the dura, owing to the slightly increased resistance to the point of the needle; the stylet is now withdrawn

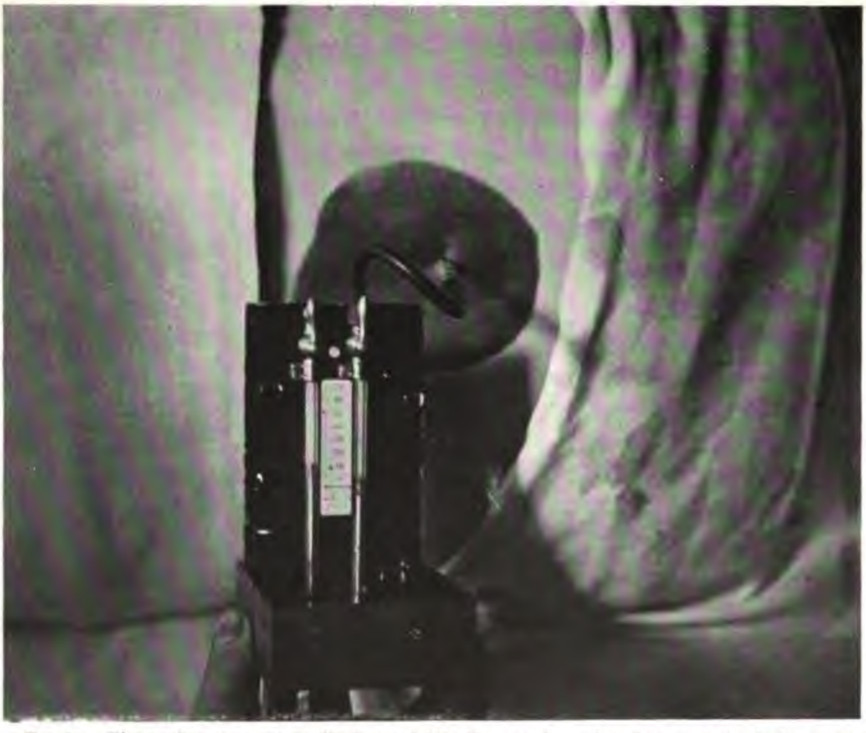

FIG. 16.—The lumbar puncture needle inserted into the spinal canal and connected with the manometer; the spinal pressure registers in this patient a height of 12 mm. of mercury. The patient is lying quietly upon the left side—the median line of the head being upon a level with the spinal canal. The skin over the area of the insertion of the needle, between the 4th and 5th lumbar vertebræ, has been painted with iodine for the asepsis.

—great care being used not to touch its point or shaft or to lay it down, and in this manner, if the cerebrospinal fluid does not flow from the needle, then the stylet may be replaced safely and the puncture needle inserted farther or moved gently up and down and laterally until the fluid appears at its lumen; it is possible at times for a small piece of tissue or a nerve filament of the cauda equina to block the inner lumen of the needle by the suction of the cerebrospinal fluid as it begins to flow through the needle, and in this manner the cerebrospinal fluid is prevented from escaping; gently moving the needle, however, usually suffices to remove this blockage. If one of the plexus of veins within the spinal canal is penetrated, blood may escape from the needle and continue to flow until the needle is blocked; it is usually wiser to attempt another puncture by withdrawing the needle, not out from the skin, but only from the dural sac and then reinserting it at another point; very frequently clear cerebrospinal fluid may thus be obtained. In some cases, however, when it is doubtful whether there is really free blood in the cerebrospinal fluid or not, it is usually wiser to perform another lumbar puncture several hours later. The important observation to be obtained in these traumatic patients, however, is the presence or not of a marked increase of the intracranial pressure and the appearance of free blood in the cerebrospinal fluid is really of little importance—especially in so far as the treatment of the patient is concerned.

The dural sac having been pierced by the puncture needle, the stylet is now withdrawn and the stop-cock turned so that the cerebrospinal fluid can flow into the sterilized rubber tubing connecting the puncture needle with the mercurial U-tube of the spinal manometer. With the patient lying perfectly quiet, the median line of the head being upon a level with the spinal canal, and the zero reading of the mercury of the manometer at a level with the spinal canal, the pressure of the cerebrospinal fluid can now be registered—the lowest level of fluctuation of the mercury being considered the pressure. If the patient is perfectly quiet with normal regular respiration, then there is practically no corresponding rise and fall of the mercury reading, but if the patient should be struggling or crying, as in children, then the reading of the manometer should not be recorded until the patient is as quiet as possible—and then the lowest level of the mercury considered as representing the approximate pressure of the cerebrospinal fluid. Under a general anesthetic, if the patient is not perfectly quiet and breathing normally, the pressure of the cerebrospinal fluid as registered by the spinal mercurial manometer may be increased 2–5 mm., and this should be deducted from the lowest reading of the mercurial level.

The normal pressure of the cerebrospinal fluid as registered at lumbar puncture by the spinal mercurial manometer varies from 5–9 mm. in adults and 4–8 mm. in children (approximately 16–30 drops per minute); 1 or 2 mm. has been obtained in patients in severe shock, and frequently in children having an agenesis or lack of development of the brain associated or not with the condition of microcephalus.

After the pressure of the cerebrospinal fluid has been accurately recorded, if it is desired (and it usually is) to remove a small quantity of the fluid for examination (Wassermann test, cell count, globulin content and colloidal gold reaction), then the rubber tube can be easily detached from the manometer and the cerebrospinal fluid allowed to flow slowly into one or more sterile test-tubes. Not more than 5 c.c. should, as a rule, be withdrawn—and particularly is this true in patients having a very high pressure due to intracranial tumor formation or large intracranial hemorrhage; the danger of the medulla being forced down into the foramen magnum and directly compressed and "collared," as it were, by the sudden lowering of the pressure of the spinal canal should always be remembered; this risk is slight and never occurs if the lumbar puncture is properly performed—that is, not more than 5 c.c. being removed (and no larger quantity is ever necessary to be withdrawn for examination) and the fluid being permitted to escape very slowly and not rapidly in spurts; firm compression upon the rubber tube easily regulates the rate of flow. When these two precautions are observed these medullary complications never occur as a result of lumbar puncture; during the past seven years in this department and clinic, over 1600 lumbar punctures were performed, and in only three patients did this medullary complication happen, and in each case the lumbar puncture was performed by an inexperienced interne who held the erroneous belief that the purpose of the puncture was to remove as much of the cerebrospinal fluid as possible and in this manner lower the extreme intracranial pressure; two of these patients had an intracranial tumor (one being subtentorial) and the third patient having the condition of internal hydrocephalus; each of these patients died within six hours after the lumbar puncture from the signs of acute medullary compression and an autopsy revealed in each case the direct medullary compression of the surrounding collar of the foramen magnum, and thus constricting the medulla in its middle portion; upon post-mortem hardening the medulla *in situ*, the characteristic furrow and groove of the compressed rim of the foramen magnum was easily demonstrable. If subtentorial lesions are excluded and the intracranial pressure is not high, then there is little or no danger in allowing a large quantity of cerebrospinal fluid to escape; this is not necessary nor advisable for diagnostic purposes, and it is only as a therapeutic measure in mild cases of cerebral edema ("wet" brain) following brain injuries and toxic and infectious conditions, such as chorea, delirium tremens, uremia and diabetic cases and in the preliminary stages of meningitis and meningeal irritation (meningismus); in these conditions, frequently repeated lumbar punctures with the removal of a larger quantity of cerebrospinal fluid each time will usually lessen, if only temporarily, the severity of the intracranial symptoms and signs and may thus facilitate the recovery of the patient. The value of these therapeutic lumbar punctures, however, has not definitely been established in these conditions, since the number of cases reported is comparatively small and not conclusive.

Upon withdrawing the puncture needle, the area of skin painted with iodine solution is rubbed with alcohol and a small sterile gauze pad applied and held in place by two small strips of adhesive plaster. It is usually advisable for ambulatory adult patients to remain in bed for at least twelve hours following the lumbar puncture; naturally, patients having acute brain injuries are confined to their bed, but it is not very exceptional for them to be walking about or to be desirous of even being out of bed. In children, however, unless the intracranial condition is an acute one, it is not so essential for them to remain in bed longer than several hours at most. The ideal time for performing the test is at night, so that the patient may sleep as usual and by morning any symptomatic effects of the puncture have usually disappeared.

The headache of greater or less severity which may follow a lumbar puncture usually occurs in the presence of an increased intracranial pressure and when more than 5 c.c. of the cerebrospinal fluid have been rapidly removed, and only occasionally if no fluid or, at most, less than 5 c.c. have been carefully and slowly withdrawn. It does, however, occur at times even when no fluid has been allowed to escape and when the puncture was made merely to estimate the degree of pressure; the various explanations for this so-called "lumbar puncture headache" are not entirely satisfactory; whether it results from a meningeal irritation, an intracranial circulatory disturbance, a temporary increase of the secretion of the cerebrospinal fluid or a continued leakage of the fluid through the puncture opening of the spinal dura (McRoberts), and thus tending to produce a "dry" condition of the brain and to permit its resting upon the base of the skull, is not definitely known.

The purpose of the lumbar puncture is two-fold: first, as an aid in the diagnosis of the cerebrospinal lesion, and second, as a therapeutic measure in selected conditions affecting the cerebrospinal system. As a diagnostic aid the lumbar puncture makes it possible for the pressure of the cerebrospinal fluid to be recorded accurately by the spinal mercurial manometer, and thus the degree of intracranial pressure be ascertained for numerous lesions, and a small quantity of the fluid itself can also be removed at the same time for the various laboratory tests, such as the Wassermann (luetic infections), cell count (acute and chronic infectious and inflammatory conditions), globulin content (tumor formations), colloidal gold reaction (paresis), etc. It is important to ascertain the degree of increased intracranial pressure in the following conditions, in order that the approximate treatment be instituted as early as possible: acute and chronic brain injuries in adults, children and new-born babies, suspected intracranial tumors, hydrocephalus of either the internal or the external type, and the numerous toxic and infectious cerebrospinal conditions, especially meningitis in its various forms, and the severe type of cerebral edema occurring in uremia, diabetes and alcoholism.

The therapeutic value of repeated lumbar punctures and removal of varying amounts of the cerebrospinal fluid in intracranial conditions associated with cerebral edema ("wet" brain) has not been definitely established; in this series of brain injuries, with and without a fracture of the skull, there has been a number of patients in whom the intracranial pressure has been increased with and without the presence of free blood in the cerebrospinal fluid and yet the pressure was not sufficiently high to warrant the cranial operation of decompression and drainage; yet, in order to diminish this increased intracranial pressure, lessen the symptoms of headache, dizziness and nausea, and the signs of stupor, restlessness, and thus improve the general condition of the patient and facilitate an early and complete recovery, repeated daily lumbar punctures with removal of 15–20 c.c. of cerebrospinal fluid were performed, and it was very impressive to observe in each of these selected patients an almost immediate improvement; usually lumbar punctures upon four or five successive days were necessary in order to lessen the pressure permanently—the patient each time experiencing such a marked relief of headache and dizziness that the slight momentary pain of the lumbar puncture itself was welcomed because, as the patient so frequently reiterated, "I feel so much better almost immediately." It was also interesting and instructive to observe the lowering of the pressure of the cerebrospinal fluid which was usually registered before it was permitted to escape, being 12, 14, or even 16 mm. of mercury, and then after withdrawing 15–20 c.c. the pressure now might be only 8, 10 or 12 mm., respectively; at the same time, the ophthalmoscopic examination before the lumbar puncture and revealing dilated retinal veins and an edematous blurring of the nasal margins of the optic disks, would frequently, within four hours after the puncture and removal of 15–20 c.c. of cerebrospinal fluid, disclose almost clear and distinct nasal margins of the optic disks and the retinal veins less dilated than before the puncture; this lessening of the intracranial pressure as revealed by the ophthalmoscope rarely continued longer than ten or twelve hours, when the retinal veins gradually became more and more enlarged while the nasal margins of both optic disks became less distinct until they were finally obscured entirely by the edema; at times even the temporal margins were blurred for several hours as though the removal of the cerebrospinal fluid at lumbar puncture had temporarily at least resulted in an increase of the intracranial pressure by stimulating the secretion of the cerebrospinal fluid; this latter observation, however, was made in only a small number of the patients. This method of lessening the increased intracranial pressure following brain injuries should, however, only be used in selected patients, in whom the pressure of the cerebrospinal fluid, as revealed by the ophthalmoscope and the spinal mercurial manometer, is known to be only mildly increased; it would be distinctly dangerous as well as inadequate for those patients having a high intracranial pressure if more than the nasal halves of the optic disks were obscured by edema and the spinal mercurial manometer registered a pressure of the cerebrospinal fluid being above 16 mm.; in these patients repeated lumbar punctures and removal of large amounts of cerebrospinal fluid might cause an immediate medullary compression and even a medullary edema be precipitated as the result of direct pressure of the rim of the foramen magnum; as a rule, not more than 5 c.c. of cerebrospinal fluid should be removed at lumbar puncture for diagnostic purposes and no therapeutic attempt made to lessen the intracranial pressure if the ophthalmoscopic and spinal manometric tests have disclosed a high intracranial pressure.

The lumbar puncture is of the greatest diagnostic value as an aid in differentiating the various kinds of cerebral spastic paralysis occurring in children. It is now definitely established that the condition of cerebral spastic paralysis with and without marked mental impairment and commonly known as Little's disease, is due to one of three causes: first, a lack of development of the cerebral tissues, and naturally there can be no increase of the intracranial pressure, which is always ascertained to be negative by the ophthalmoscopic and spinal manometric tests and therefore not amenable to any cranial operative procedure; secondly, a meningitis and meningo-encephalitis—a destructive process of varying degree of the cortical nerve cells and not to be benefited by an cranial operative procedure, unless the intracranial pressure is markedly increased as a result of a secondary external hydrocephalus due to the blockage of the stomata of exit of the cerebrospinal fluid, and in these latter selected patients only may the condition be improved and the associated convulsions lessened by the operation of cranial decompression and drainage; according to the degree of blockage of the cerebrospinal fluid and therefore of the resulting external hydrocephalus are the signs of an increased intracranial pressure revealed by the ophthalmoscopic and spinal manometric tests which, together with the history and associated or not with convulsive seizures, form a fairly typical picture; those patients in the group due to toxic and infectious conditions producing cerebral thrombi and emboli naturally do not cause an increase of the intracranial pressure since a localized cerebral atrophy results; and lastly, and a most common cause of cerebral spastic paralysis in children is an intracranial hemorrhage at or near the time of birth; the hemorrhage is almost always a supracortical one—rarely within the cortex itself—and thus the cortical nerve cells are not destroyed but merely functionally impaired by the pressure of the overlying hemorrhage and the development of a secondary external hydrocephalus as a result of the blockage of the stomata of exit of the cerebrospinal fluid in the cortical veins, sinuses, etc.; these are the patients having an increased intracranial pressure as revealed by the ophthalmoscope and the spinal mercurial manometer, whose history is most frequently one of difficult labor at a full-term birth with and without the use of instruments and associated or not with convulsive seizures; they are usually first children; unless an early lumbar puncture is performed to ascertain the presence of blood and an increased pressure and the true condition of the brain injury recognized, the spasticity of the legs or arms may not be observed until the child is seven and even nine months of age; the child is retarded both mentally and physically—does not hold its head up nor attempt to sit up or walk until months after it should, and at the same time the mentality is delayed so that these children become not only physically but mentally impaired. The ideal time for the relief of the increased intracranial pressure in these patients is as soon as possible after birth—when the hemorrhage can be drained in fluid form either by repeated lumbar punctures daily or by the cranial decompression and drainage; if the true intracranial condition is not recognized within several days after birth, then the decompression and drainage should be performed as early as possible in order to lessen the cortical compression and thereby permit a normal cerebral development both mentally and physically.

CHAPTER VII

THE TREATMENT OF BRAIN INJURIES WITH AND WITHOUT A FRACTURE OF THE SKULL

General Considerations.—For many years, the routine treatment for brain injuries, or rather the so-called "fractures of the skull," whether of the base or of the vault, has been the expectant palliative one; that is, an ice-bag to the head, vigorous catharsis, liquid diet, and absolute rest and quiet, morphia being administered if necessary. Practically all patients having fractures of the base were thus treated—it being thought that nothing else could be done for such patients; the mortality was high—more than 50 per cent. Even depressed fractures of the vault, unless there were localized signs of cerebral compression, were frequently treated in the same manner.

Naturally, the patients having simple concussion and the mild conditions of brain injuries with and without a fracture of the skull—and I believe that many cases of fracture of the skull are overlooked on account of their comparatively trivial symptoms and signs—have been, and are being, treated successfully by this expectant palliative method; it is, however, in those patients having brain injuries with and without a fracture of the skull, whether of the base or of the vault and with or without a depression of fragments, in whom there are marked signs of an increased intracranial pressure that this expectant palliative treatment is not sufficient, and a more effective method of lowering this increased intracranial pressure is essential.

Within the last few years, a notable advance has been made in the treatment of these patients. It is not so much a question of ascertaining the presence and site of the fracture (unless it is a depressed fracture of the vault), but rather of finding out whether there is or is not an increased intracranial pressure, and if there is, then directing the treatment toward a lowering of this abnormal pressure. For this reason the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer should be registered, and also careful ophthalmoscopic examinations should be made in each patient in order to observe the early signs of an increased intracranial pressure appearing in the fundi of the eyes, and especially about the entrance of the optic nerve—the so-called optic disk. These changes in the fundi are the result of increased intracranial pressure, whether this pressure be due to a slowly growing tumor, to an intracranial hemorrhage, or to a very edematous "swollen" brain resulting from a brain injury with and without a fracture of the skull. It is this cerebral edema, resulting in varying degrees from any injury to the brain—from the mild conditions of concussion to the worst forms of cerebral contusion and laceration—that has been overlooked in the past; in my opinion, it is the most important factor to be considered in any cranial injury. In mild cases only a slight dilatation of the retinal veins results from the intracranial pressure due to this cerebral edema, and the veins gradually assume their normal size and appearance within two to four days, showing that the intracranial pressure has been lessened as the result of the absorption of the edema; that is, there is present now a more normal amount of intracranial cerebrospinal fluid.

The success of the expectant palliative treatment in these mild cases (and they form about two-thirds of the patients having brain injuries) is based upon the fact that, by it, not only is the absorption of the cerebrospinal fluid increased, but the amount of cerebrospinal fluid secreted is lessened by the lowering of the blood-pressure and thus the tendency to intracranial hemorrhage is also lessened. Naturally, the sooner after the injury the treatment is started the better are the results to be obtained.

In the more severe cases, however, a more direct method of lowering this increased pressure is necessary; I use the word "severe" not so much in reference to the fracture as to the height of the intracranial pressure. These are the patients showing not only a dilatation of the retinal veins and an edematous obscuration of the nasal margins of the optic disks, but also a blurring of the nasal halves of the optic disks and the more advanced signs of intracranial pressure to be observed with an ophthalmoscope (although it is rare for "choked disks" to be observed in these patients), and the spinal mercurial manometer registers the pressure of the cerebrospinal fluid above 15 mm. Such patients should have a subtemporal decompression and drainage performed to relieve the intracranial pressure as soon as possible after the injury. If, however, the patient is in a condition of severe shock, so that its signs prevent, overshadow and tend even to conceal the signs of intracranial pressure, then all treatment should be directed toward relieving the condition of shock, and when this has been accomplished, then the lowering of the intracranial pressure can be considered; it is neither wise nor good surgical judgment to decompress a patient having a brain injury and that patient is in severe shock, with a pulse-rate of 110 or more; if the patient is unable to survive the shock, surely no operation will aid him but will be merely an added shock. The operation itself is not a formidable one; naturally, perfect asepsis is essential. The anesthetic should be administered by an expert; only too frequently, however, the patient is unconscious, so that an anesthetic is not required.

Palliative *versus* Operative Treatment.—In too many hospitals the attitude toward patients having brain injuries and the so-called "fractures of the skull," and especially those of the base, has been one of expectancy, a policy of "letting well enough alone"; unless there was a marked depressed fracture of the vault and the signs of local cortical compression, then the usual treatment of these patients was an ice-bag to the head, catharsis, absolute rest and quiet, and the general routine expectant palliative treatment was administered; any operative procedure was not to be considered unless the patient developed signs of compression of the medulla—slow pulse of 50 and lower, irregular respiration of the Cheyne-Stokes type and a blood-pressure of 170 and higher. Then and only then would an operation be advised and performed, and with the usual result—the death of the patient. Several days may elapse before the signs of extreme medullary compression occur, but once the later signs of medullary edema and collapse do appear, then a cranial operation will be of no benefit unless to possibly 1 per cent. of the patients—the others all die, operation or no operation. The time for the operation of decompression and drainage should be judged according to the general condition of the patient and the amount of intracranial pressure as shown by the ophthalmoscope and the spinal mercurial manometer, and not by the extreme signs of medullary compression, such as a very low pulse- and respiration-rate and a high blood-pressure. The old method of not operating until definite signs of medullary compression occurred—that is, the signs resulting from the extreme degree of increased intracranial pressure—accounts for the high mortality of operations performed at this late period, and justified the opinion of so many observers in the past that the expectant palliative treatment of "fractures of the skull" is equally successful as the operative treatment. Patients, however, should not be permitted to reach this dangerous condition of medullary compression, as its cause and forerunner, high intracranial pressure, can now always be revealed by the routine use of the ophthalmoscope and accurately measured by the spinal mercurial manometer. Besides, an early decompression and drainage will not only save the lives of a large percentage of patients who otherwise would have died from medullary compression, but it will lessen the percentage of post-traumatic conditions so frequently following brain injuries with and without a fracture of the skull: in the mild cases, those vague indefinite headaches, associated at times with dizziness, a throbbing sensation in the head, and the early signs of fatigue, so commonly observed and considered as "post-traumatic neuroses"; in the more severe cases, a complete change of personality—the patient becoming either very irritable and restless, indulging in fits of anger at the least provocation, and having so little self-control that he is unable to hold any position permanently; or the reverse, very much depressed, with loss of ambition, a "happy-go-lucky," and in many patients, as the relatives have expressed it, a "bum" and "good-for-nothing"; epilepsy, especially in the minor form of "*petit mal,*" and at times "*grand mal*"—is fairly common after depressed fractures of the vault, but fortunately it is more rare following basal fractures, due, possibly, to the resulting hemorrhage being more at the base and not over the cerebral cortex.

Such has been the record of my following the histories of patients who have had brain injuries and the so-called "fractures of the skull," particularly of the base, and have been treated by the expectant palliative treatment in three of the large hospitals in New York City during the decade of 1900–1910. These patients remained in the hospital for periods of two to six weeks, and were almost without exception discharged as "well" or "cured." Naturally, it has been a most difficult undertaking to locate these patients, especially after a lapse of five years and more, and particularly since the vast majority of them were the usual ambulance patients of the poorer classes who change their residences almost as frequently as the seasons come and go; my inability to locate more than 34 per cent. of them may thus be explained. The most striking thing, however, is that, of the patients found, 67 per cent. are still suffering from the effects of the brain injury and the "fracture of the skull"; that is, they have not had the same good health since the accident as before—the most frequent complaints being headaches of greater or less severity, changes of personality of the exalted and of the depressed types, a nervous instability, and occasionally epilepsy in its various forms; that is, about two-thirds of the patients whom I was able to locate were not well. It is for this reason, no doubt, that it is popularly believed that "once a person has had a fracture of the skull, he is never the same person again"; these statistics would tend to confirm this belief.

The patients operated upon in these three hospitals were chiefly ones having depressed fractures of the vault; upon those operated patients having "fractures of the base of the skull," the operation was performed only upon the ones showing signs of definitely localized cortical compression and of marked medullary compression and even edema itself, and naturally the mortality was very high—being 87 per cent. Besides, an extensive bone-flap operation was frequently performed and the bone-flap then replaced, thus lessening and even preventing the benefits of a decompression; in some patients, the dura was not opened, and, therefore, the benefits of even a limited decompression could not be obtained, because the dura is inelastic in adults, and it always must be opened if a decompression is desired; simply removing an area of bone is not a decompression.

Of those patients having "fractures of the base" who were operated upon at the Johns Hopkins Hospital since 1900, 58 per cent. were located in 1913, and of this number 32 per cent. were suffering from the effects of the cranial injury; of the patients operated upon since 1906, only 22 per cent. were still impaired, due undoubtedly to an earlier operative interference.

My experience in hospitals of Boston and New York City, where the expectant palliative treatment was adhered to in "fractures of the base," and then later at the Johns Hopkins Hospital, where selected patients having a high intracranial pressure were operated upon comparatively early, strongly impressed me with the superiority of the latter treatment. During the past six years, I have advised the cranial operation of subtemporal decompression and drainage upon patients having brain injuries with and without a fracture of the skull as soon as definite signs of a marked increase of the intracranial pressure can be demonstrated by the ophthalmoscope and confirmed by the spinal mercurial manometer, and the results have been most gratifying; not only is the danger of a medullary edema lessened by an early operation and an immediate relief of the intracranial pressure obtained and thus a higher percentage of recovery of life, but the number of the post-traumatic conditions, both physical and mental, has been very much diminished—to less than 14 per cent.

In this series of 155 operated patients (to January 1, 1919), the percentage of these patients still suffering from the effects of the brain injury with and without a fracture of the skull is 13.4 per cent.; these were the extreme cases having cortical lacerations and numerous small hemorrhages in the cortex, as revealed at the operation. Naturally, sufficient time has not yet elapsed to render these figures regarding the post-traumatic conditions of the greatest value, and it will be necessary to wait at least five years longer in order to obtain more accurate data regarding them. For fear of being misunderstood, however, I wish to repeat that the cranial operation of subtemporal decompression and drainage is only for selected patients having brain injuries with and without a fracture of the skull—only in the ones showing marked signs of an increased intracranial pressure and comprising about one-third of the patients; whereas the expectant palliative method of treatment alone is sufficient for almost two-thirds of the patients—the ones having no marked signs of an increased intracranial pressure. Fortunately, it is a fairly frequent occurrence to have a fracture of the skull (confirmed clinically and by the X-ray) with no marked signs of intracranial pressure; in these patients a cranial decompression can do no good and would be only an added risk. But, in patients showing marked signs of increased intracranial pressure, the early relief of this pressure is essential, not only to lessen the percentage of the post-traumatic conditions, but to avoid a medullary edema and its resulting high mortality.

A. **Expectant Palliative Treatment.**—*Shock.* The presence of shock in varying degree is a factor in over four-fifths of the patients having acute brain injuries, so that its appropriate treatment is of the most urgent consideration; the extreme shock must first be overcome and disappear early if the patient is to survive; and unless this initial period of severe shock subsides, then the local cranial and intracranial examination and treatment must be deferred in the hope that the patient will react to the vigorous measures used to combat the shock. Almost 10 per cent. of the patients having severe brain injuries are unable to recover from this initial condition of extreme shock—frequently due to the absence and inefficiency of the proper shock measures being instituted as soon as possible following the cranial injury: the patient is permitted to lie upon the cold ground, pavement or floor until the ambulance arrives—a period of time varying from thirty minutes to one hour and even longer; a blanket may be thrown over the patient but rarely is he wrapped in blankets. (I am describing the usual "first aid" treatment afforded these patients by the policemen and onlookers following a typical accident in the streets of the city whereby the patient has been struck by an automobile, street car or subway train, or has been injured in an industrial accident.) An ambulance is summoned by the policeman, and everyone waits until the ambulance arrives—the patient receiving scant attention other than the "keeping-back" of the crowd, "give the man plenty of air," and so frequently the attempt to administer whiskey by mouth to an unconscious, or at most, a semi-conscious man. If, while awaiting the ambulance, the patient could be lifted from the ground or the floor to a bench, a counter or even a couch and bed, warmly wrapped in overcoats and even heated blankets, with several "hot" water bottles applied to the extremities and body, and no attempts made to waken and arouse the patient but rather "let him alone," it is my opinion that a larger number of these patients would survive this initial period of severe shock. Lying upon the ground and floor and inadequately wrapped, these patients are "chilled through" in addition to the shock of the cranial injury itself, and thus they undergo an exposure which ordinarily would produce a "cold" even in the presence of good health. The immediate "putting-to-bed" of these patients in heated blankets is rarely possible on account of the location of the accident.

Upon the arrival of the ambulance, however, the patient should then at least be warmly wrapped in heated blankets (and it is rare for a hospital ambulance to have them, or even "hot" water bags or electrically heated stoves as now in very common use in a limousine in winter); the use of drugs hypodermically is rarely of great value in this stage of severe shock, although camphor in oil, caffeine, atropine and strychnia may be administered; codeine or morphia are excellent if the patient is conscious or restless. It is, however, the early routine use of vigorous external heat—best by means of heated blankets—and then keeping the patient as quiet and free from disturbing examinations, that these unconscious and semi-conscious patients are most benefited and assisted in combating the condition of shock. Care should be taken by the driver of the ambulance not to "bounce" and jar the patient during a "fast" ride to the hospital; it has happened several times in my experience for the ambulance itself, under these conditions, to have accidents on the way to the hospital so that the original patient with the brain injury was joined by another patient similarly injured by the ambulance itself, and in one instance, by two patients. These "fast" and reckless ambulance trips through the city streets rarely if ever aid the patient being transported; a delay of five or ten minutes, but in safety, is in no way harmful to a patient in an ambulance and attended by a competent doctor.

Upon arriving in the ambulance at the hospital and in the condition of severe shock, if the patient can be placed immediately in a bed between heated blankets and with several "hot" water bags to the extremities and body and then not disturbed by examinations but permitted to remain in absolute quiet—that patient is indeed a most fortunate one. These patients in severe shock and having a pulse-rate over 110 should be treated for the shock alone: first and most important, external warmth, absolute quiet—morphia being administered freely in order to obtain it—and rectal enemata of hot black coffee in amounts of two to four ounces every two to four hours; camphor in oil hypodermically is excellent. Merely a superficial examination upon admission is sufficient in order to ascertain the presence or not of fractures of the extremities, internal injuries of the abdomen and chest, and to arrest any profuse hemorrhage; scalp wounds should be widely shaved, thoroughly cleansed and loosely sutured, but to examine carefully and repeatedly the reflexes, to make ophthalmoscopic examinations and even a lumbar puncture in this stage of shock is meddlesome and of no value to the patient—in fact, a probable harm to the patient in prolonging and even increasing the severity of the shock. It does not benefit either the patient, in this stage of shock, or the doctor to ascertain the presence or not of a fracture of the skull, any inequality of the reflexes or the presence of blood in the cerebrospinal fluid—these data can be elicited after the shock has lessened or even entirely disappeared and then the appropriate treatment vigorously instituted, without any danger of producing shock, and in this manner the patient is afforded the best chance of immediate and ultimate recovery.

The history of these patients in most hospitals unfortunately is the following: The patient is carried upon a stretcher from the ambulance into the accident-room of the hospital and placed upon an examining table—covered with padded leather frequently, but only too often not covered and not soft; a blanket may or may not be placed over the patient and it is rarely, if ever, a heated and warm one. The interne of the hospital staff assigned to duty in the accident-room is now summoned; upon arriving, he obtains the history of the patient from the ambulance doctor and examines the patient to determine whether the patient is severely enough injured to warrant the summoning of the resident house-surgeon to decide whether the patient should be admitted to the hospital ward or not; upon his arrival, which may be delayed a number of minutes, and then after his examination, the patient is transferred to the ward or a private room and placed in bed—and naturally the bed should be warmed with "hot" water bags and the patient wrapped in two or more heated blankets. It will be seen, however, that the usual hospital patient has not only been not warmed and vigorously treated for this condition of shock, but that he has been repeatedly examined—at the time of the injury, by the ambulance doctor, the accident-room doctor, the resident surgeon and finally sometimes even by the attending surgeon to the hospital; a period of time varying from over one hour to even three hours may be consumed before the patient reaches a bed and is really warmed in the treatment of the shock. It is this delay in the appropriate treatment of many of these severely shocked patients that the condition of shock is prolonged, and only too frequently increased, so that the patient is unable to survive this extreme degree of shock; these patients at autopsy may or may not reveal a fracture of the skull, and may, but usually they do not, disclose a large intracranial hemorrhage or an increased intracranial pressure due to cerebral edema; the usual post-mortem findings are merely an anemic, pale condition of the brain and, even in the presence of rather large cortical vessels being torn, yet the amount of free hemorrhage is small, showing that the lowered blood-pressure of shock had not been of sufficient force to overcome the normal intracranial pressure and thus cause an intracranial hemorrhage; if the shock had lessened, then the general blood-pressure would have increased so that it would have been possible in these patients for a large intracranial hemorrhage to occur and later its resulting high intracranial pressure.

It is not a question in these patients in severe shock with a pulse-rate over 110 whether the skull is fractured or not or how badly, or whether there might be an intracranial hemorrhage or not, or whether it appears the patient is going to die: all efforts should be directed toward the lessening of the extreme condition of shock by raising the general lowered blood-pressure of shock—best accomplished by external warmth, absolute quiet, repeated rectal enemata of hot black coffee and the use of camphor in oil, caffeine, atropine or strychnia; camphor in oil, hypodermically, has been, in our experience, possibly of greater value than any other drug for the condition of shock, although hot black coffee by rectum is almost of equal value.

The value of morphia for restlessness and also for the shock in many of these patients cannot be overestimated. Formerly, in conditions of brain injuries, the use of morphia was condemned for fear the drug would mask the symptoms and signs of an increasing intracranial hemorrhage and therefore, the appropriate operative treatment be delayed and even omitted until the terminal stage of the condition. This criticism of the use of morphia was perfectly well justified several years ago, when the symptoms and signs of an intracranial lesion indicative of the necessity of operative interference depended almost entirely upon the presence or not of paralyses, inequality of the pupils, a low and irregular pulse- and respiration-rate and an increased blood-pressure; these signs, however, are now known to be most crude ones of the terminal stages of high intracranial pressure producing a medullary compression, whereas the use of the ophthalmoscope, and especially the spinal mercurial manometer, has made it possible to ascertain accurately the intracranial status of the intracranial pressure—whether due to hemorrhage or edema—and thus the use of morphia can in no way lessen or impair the value of the examinations. Morphia will produce a pupillary constriction but rarely a narrowing so small that a careful ophthalmoscopic examination cannot be made, and if it should, then the most accurate measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer will still be possible.

If the severity of the extreme condition of shock lessens so that the pulse-rate gradually descends from 130 or more down to 100 and lower, and the blood-pressure ascends from 100 or lower up to 120 and higher, then a thorough neurologic examination becomes possible and yet the patient is in no way harmed by increasing or prolonging this mild condition of shock.

As a routine method of treatment, the following measures are now important:

I. *Absolute Rest in Bed, Quiet and Warmth.* The room should be cool and darkened, and there should be the greatest possible freedom from noise and from disturbing elements. Relatives should be excluded from the sick-room unless the patient is unconscious, and even then it is a wise measure, and most assuredly if the patient is conscious, in order that the emotions be not aroused. Small repeated hypodermic injections of morphia (gr. 1/8) are most useful for insuring quietness to restless, excitable and even delirious patients. The head should not be elevated beyond the height of one pillow, and frequently it is advisable not to raise the head at all, and even to lower it in the conditions of extreme shock by elevating the foot of the bed; warm blankets and "not too warm" hot-water bags should be applied to the body, and even flannel or rubber bandages may be wrapped about the legs and also the arms. In conditions complicated by alcoholism, an immediate gastric lavage is beneficial. The patient should remain quietly in bed for at least a period of ten days to two weeks. All reading should be prohibited; many "nervous breakdowns" following brain injuries result from the neglect of this simple precaution. The patient should not attempt to return to active business for at least a period of three months, and better, six months.

II. *Catharsis.* Upon the subsidence of the severe shock, an enema of soapsuds or oil should be given, and if the patient is conscious, then a cathartic administered by mouth—either a saline or calomel in 1/2 gr. doses, to be followed by a saline purge. Vigorous catharsis is very important, and yet in conditions of severe shock it should be delayed until the patient is recovering from the shock; naturally, if the patient is unconscious, then enemata only should be used. In the hospital, it is a routine procedure to give a soapsuds enema each morning for at least two weeks after the injury; in these patients, the blood-pressure has been frequently observed to descend ten to fifteen points following a soapsuds enema with a large movement of the bowels.

III. *Cold Compresses to the Head.* After the severe condition of shock has disappeared, a large ice-bag (ice-helmet) surrounding the entire head should be applied. The coldness of the ice tends in a small way to lessen the cerebral circulation and thus diminish and even prevent intracranial hemorrhage and the cerebral edema resulting so frequently from injuries to the head; in this manner, a lowered intracranial pressure is obtained. The ice-bag should not be allowed to remain about the head for periods longer than one hour; after an interval of one-half hour, it can be replaced for another period of one hour, and so on. Patients will frequently ask for the ice-bag to be replaced—it so relieves the "throbbing" in the head and the headache, and the request should always be granted, as the patient is an excellent judge of its efficacy; the patient will not ask for the reapplication of the ice-bag if there is neither throbbing nor headache to be lessened.

IV. *Diet.* For unconscious patients nothing by mouth, naturally, should be given; after two days, these patients will usually regain consciousness sufficiently in order to swallow; if not, then nutrient enemata may be employed. A liquid diet should be adhered to for several days; any liquid food may be given, although milk and its modifications should be avoided for at least three days after the injury, for fear of the formation of gas and the resulting abdominal discomfort. Alcohol in any form whatsoever should be avoided; however, if the patient is alcoholic, it is wise to administer at least one-half ounce of whiskey or brandy three times a day for fear of the possible onset of delirium tremens. After the patient's discharge from the hospital, red meats and meat soups should be banned for a period of three and, better, six months or a year; the danger of increasing the irritability of the cerebral cortex must always be remembered; especially is the use of alcohol in any form whatsoever to be prohibited, and for years.

V. *Drugs.* Except for the use of morphia in conditions of extreme restlessness and shock, and camphor in oil and hot black coffee per rectum as a routine procedure, there are few drugs worth mentioning in the treatment of brain injuries with and without a fracture of the skull. Strychnia may be given in conditions of shock, but its real value is doubtful. Most important, however, in conditions of shock of varying degree is hot black coffee given slowly by rectum in amounts of four to eight ounces; I have frequently seen remarkable improvement in the general condition of these patients after its administration and also following cranial operations of any great severity, when its value is of the greatest importance. Atropine in repeated doses of gr. $^{1}/_{60}$ is of the greatest value in conditions of early pulmonary edema—a much-dreaded complication in the more seriously injured patients during either the period of initial shock or the terminal one of medullary edema. The routine treatment, as briefly outlined above, should be followed in all patients having a severe cranial injury—that is, an effort should be directed toward the prevention and lessening of an increased blood-pressure, and in this manner it is possible to lower, and even avoid, the formation of an increased intracranial pressure. Naturally, if the symptoms and signs of initial shock are the more prominent, then the treatment should be directed toward the relief of the condition of severe shock, and when this has been accomplished, then the intracranial condition can be considered.

If in a condition of extreme shock, then the patient should not be disturbed—not even for the purpose of making an examination. It will not benefit him, and in some severe conditions it may do him harm; the treatment remains the same as outlined above whether the patient has a fracture of the skull or not, and for this reason a thorough physical and neurologic examination should be deferred until a definite improvement in the general condition of the patient and especially in the severity of the shock has been obtained.

VI. *Aseptic Measures.* It is of the greatest importance in all lacerations of the scalp, and even in severe contusions, to shave carefully the surrounding area—at least one inch beyond the margin of the laceration; to remove all foreign bodies with scrupulous care, cleanse the wound thoroughly with green soap, and then carefully apply alcohol or a weak solution of iodine to the damaged tissues. If the underlying vault is fractured and there is a possibility of the adjacent dura being torn, then the greatest care must be used to avoid any cerebral irritation resulting from the use of the alcohol or iodine; the immediate danger of thus causing epileptiform seizures would be a most serious complication as well as an unnecessary and avoidable one. Gentle probing may be used to ascertain the true condition of the underlying bone. The scalp laceration can now be sutured loosely with any of the usual suture materials and a drain of rubber tissue inserted at each end of the wound. The danger of infection from foreign bodies, hair, dirt, etc., and a resulting meningitis, is so great that the utmost care and strictest asepsis are essential in all wounds of the scalp; only too frequently are such wounds carelessly treated—most commonly the surrounding scalp not being shaved—and the results are at times appalling.

If an extensive hematoma is present and of sufficient size that the overlying scalp is very tense and therefore less viable and less resistant to a scalp infection, it is very important to ascertain the presence or not of a fracture of the underlying vault of the skull by an early röntgenogram. If there is a line of fracture of the underlying bone, then I feel it is better surgical judgment to aspirate the blood of the hematoma through a "clean" area of the overlying scalp painted with iodine, and a firm compress and bandage applied. The great danger of these hematomata becoming infected and the easy and rapid transmission of the infection through the line of fracture intracranially, and thus a purulent meningitis and meningo-encephalitis, is a most serious complication; it has occurred in several patients of this series of brain injuries. If there is no fracture of the bone beneath the hematoma and the overlying scalp is in an excellent condition and is not badly contused, then the gradual absorption of the blood of the hematoma will usually occur without any complication; however, in very tense hematomata, and especially if the overlying scalp is bruised and even infected, then it is most important for the hematoma to be aspirated, as it is possible for the superficial scalp infection to extend intracranially from an infected hematoma through the diploetic veins; particularly is this true in the median areas of the scalp overlying the longitudinal sinus and its numerous tributaries. Besides lessening the danger of infection, the aspiration of the blood of the hematoma overlying a linear fracture of the vault facilitates the drainage of a possible extradural and of a subdural hemorrhage and edema (if the dura itself has been torn), so that by this means alone the intracranial pressure is lessened and even prevented from reaching a height necessitating the operation of cranial decompression and drainage; that is, the patient has decompressed himself through the line of fracture of the vault in a similar manner as through a basal fracture into the nose or ears—and with comparative safety and freedom of danger. Naturally, small hematomata do not render the overlying scalp so tense that the complication of infection is a probable one, and therefore they are rarely aspirated or drained unless the contiguous scalp is badly damaged and infected. It cannot be urged too strongly that the scalp of each one of these patients should be carefully shaved, scrupulously cleansed with green soap and water, and if necessary with alcohol, and sterile dressings applied; numerous complications will thus be avoided.

VII. *Lumbar Puncture Drainage as a Therapeutic Measure.* After the shock of the cranial injury has been lessened so that the pulse-rate is 100 and lower and the blood-pressure 120 and higher, it is advisable, in addition to the ophthalmoscopic findings, to ascertain accurately the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer; not only will the presence or absence of blood be demonstrated, but, of the greatest importance, the intracranial status of increased pressure or not be established and thus the advisability of continuing the expectant palliative method of treatment or, in the presence of a high intracranial pressure, the necessity of an immediate cranial operation of decompression and drainage. It is very infrequent in these early cases for the condition of high intracranial pressure to be demonstrated either by the ophthalmoscope or by the spinal mercurial manometer on account of the recent condition of severe shock, and yet if an acute cerebral edema occurs—and it frequently does, especially in patients having cardiovascular and cardio-renal disease and in those addicted to the daily use of alcohol—or if a large intracranial vessel has been torn, then as the shock subsides, the hemorrhage may be such a rapid and extensive one that the typical signs of low pulse- and respiration-rates and a high blood-pressure are frequently not possible and the condition of the patient may merge into that of medullary edema and collapse without the usual clinical signs of high intracranial pressure being disclosed. In these latter patients having an increased intracranial pressure to the height of over 16 mm. as registered by the spinal mercurial manometer, the immediate relief of this pressure, whether due to hemorrhage or edema, by means of a decompression and drainage is of less risk to the patient and offers a greater chance both of recovery of life and of ultimate normality, than the expectant palliative method of treatment.

In the majority of patients, however—and they form almost two-thirds of the total number—the intracranial pressure was not increased or only mildly so—not over 12–14 mm. as registered by the spinal mercurial manometer, and therefore the expectant palliative treatment was alone sufficient to obtain an excellent recovery. It was frequently observed in many of these patients who had an increased pressure of possibly 12–14 and even as high as 16 mm., that after the lumbar puncture was performed and possibly 10 c.c. carefully withdrawn for examination, a large percentage of them almost immediately improved in their general condition: their stupor lessened so that they became conscious, while the conscious patients who had been complaining of intense headache, restlessness and even nausea and vomiting, immediately "felt better"—the headache lessened and in a few patients it disappeared entirely, they became quiet and their nausea ceased. This improvement was only a temporary one in most of these patients, in that the complaints returned within several hours, and we were then impressed as to the advisability of performing repeated lumbar punctures upon these selected patients having only a mild increase of the intracranial pressure, not only by this temporary improvement but by the patients themselves, who in many instances asked that the "back be tapped again" and "the fluid removed."

During the past three years, these selected patients having an increased intracranial pressure but of not sufficient height to make necessary the cranial operation of decompression and drainage, have been repeatedly treated by this method of drainage at lumbar puncture—in many patients the procedure has been used even five and six times, and the results have been very gratifying; not only is the general condition of these patients improved, but their convalescence is hastened. It should be remembered, however, that it is only in the patients having a mild increase of the intracranial pressure that this method of lumbar puncture drainage should be advocated and in whom the complaints of headache, restlessness and nausea are severe; patients having a high increase of intracranial pressure—over 16 mm.—should not be subjected to the risk of withdrawal of cerebrospinal fluid in amounts of 10–20 c.c. for fear that this high intracranial pressure would force the medulla into the foramen magnum owing to the sudden lessening of the spinal pressure following the lumbar puncture, and thus the signs of a direct medullary compression would occur, and usually the early death of the patient. At the first lumbar puncture, if the pressure is high, then no fluid may be withdrawn, or at least not more than 5 c.c. should be slowly removed—merely for examination; if the pressure is not over 16 mm., then 10 c.c. can be safely removed, and if the complaints of headache, etc., return after several hours, a second lumbar puncture may be performed; if the pressure is again not over 16 mm., it is now possible to remove slowly and safely 15–20 c.c. of the fluid, and this procedure can be repeated safely as long as the pressure does not exceed 16 mm. In this manner, many of these patients who otherwise would recover slowly under the expectant palliative treatment alone and within a period of several weeks, but complaining of the severe headache, etc., will make an excellent recovery within a period of usually ten days to two weeks. Besides, in a few patients this early removal and drainage of the excess cerebrospinal fluid, with and without the presence of blood, will so lessen and prevent the formation of a high intracranial pressure that it will not be necessary to perform the operation of cranial decompression and drainage; that is, lumbar puncture drainage in these selected patients is really a spinal decompression and drainage. The improvement following each lumbar puncture drainage has usually been only a temporary one, and yet in the larger percentage of these patients the pressure registered at each sucessive puncture has frequently not been so high as at the preceding one, and after two, three or more punctures the pressure does not exceed a height of 10–11 or at most 12 mm.—and the patient no longer suffers intense headache, and his general condition is improved in every way. This method of lumbar puncture drainage is of special value to patients whose condition is complicated by chronic alcoholism, or by chronic nephritis and arteriosclerosis; the great danger of a "wet" edematous brain resulting in these patients is a most serious one and the early repeated removals of 15–20 c.c. of cerebrospinal fluid may tend to offset this most serious complication; acute delirium tremens may also thus be avoided.

B. **The Operative Treatment.**—The operative treatment of patients having brain injuries with and without a fracture of the skull is restricted to those patients only for whom the expectant palliative method is not sufficient either to obtain a recovery of life or to secure the greatest ultimate improvement, so that the later condition of the patient will approximate that of his former good health of the period before the injury. As has been stated, the expectant palliative method of treatment is entirely satisfactory and sufficient for almost two-thirds of these patients (if depressed fractures of the vault are excluded and for whom the operation of removal or elevation of the depressed bone is always advisable), whereas the cranial operation of decompression and drainage is essential in about one-third of the patients in order that not only a greater percentage of recovery of life be obtained, but that the former good health of the patient be restored to its maximum. There is a small percentage of these patients, however, who have suffered a laceration of the cerebral cortex and naturally the cortical cells and subcortical fibres involved are not regenerated, and therefore a permanent impairment of them results; fortunately, these cortical lacerations and severe cerebral contusions occur most frequently in the comparatively silent areas of the brain, such as the anterior portions of the temporo-sphenoidal lobes and of either frontal lobe, and especially their anterior superior surfaces, so that there is frequently little if any clinical evidence of the cerebral damage; if, however, the laceration should occur in the motor cortex of either parietal lobe, or in the motor speech area of the left posterior third frontal convolution (in right-handed patients) or in the definite sensory areas of the cortex, such as either parietal lobe posterior to the fissure of Rolando, the portion of either occipital lobe developed for the special sense of sight, or of either temporal lobe (and particularly of the left lobe in right-handed patients) specialized for hearing, taste, and smell, and if the laceration should extend subcortically into the fibres of the pyramidal tract above and below either internal capsule, then in these patients there persists a permanent impairment clinically of definite degree. The cortical cells and fibres adjacent to the laceration, however, may not be primarily destroyed but only functionally impaired by the compression of the associated hemorrhage and cerebral edema, so that as the hemorrhage and cerebral edema are absorbed by the natural means of absorption or the acute compression lessened in the patients having a high intracranial pressure by means of the cranial operation of decompression and drainage, then a marked improvement is frequently observed, although there always remains clinically the impairment of the cortical cells primarily destroyed by the laceration or the severe contusion.

It must always be remembered that as long as the severe condition of shock persists so that the pulse-rate is higher than 110 and surely above 120, even though there are signs of high intracranial pressure (and this is a very rare observation in the condition of severe shock), then the patient must first be allowed to recover from the extreme degree of shock before any operative procedure to lessen the increased intracranial pressure can be considered. A cranial operation in this period of severe shock merely increases or at least prolongs this condition of extreme shock—the operation itself being an added shock—and the chances of the patient being able to survive the shock alone are thus diminished. It is possible for a large hemorrhage to have occurred intracranially immediately following the injury and before the condition of extreme shock has occurred, but as long as the condition of severe shock persists, then it need no longer be feared that the intracranial hemorrhage is becoming greater, because the general blood-pressure has been so lowered by the condition of shock—to 110 and even lower—that no more bleeding intracranially is possible until this extreme condition of shock has disappeared. If the patient survives this condition of shock, then the blood-pressure will ascend and it is now that the most careful and repeated examinations are essential in order to determine whether the intracranial pressure is increased or not, or whether it is of sufficient height—not above 16 mm. as registered by the spinal mercurial manometer—in order that the expectant palliative treatment may be continued (and this method can be in about two-thirds of these patients), or whether the intracranial pressure is so high (above 16 mm.) that the operation of decompression and drainage is advisable in order to offer the patient a greater chance both of recovery of life and of future normality; about one-third of the patients having brain injuries are more rationally treated by this operative method.

It is, therefore, of the greatest importance after the extreme shock has subsided so that the pulse-rate has descended to 100 and even lower, and the blood-pressure has risen to 120 and higher, and the temperature to normal and above, that thorough neurologic examinations should be frequently made, ophthalmoscopic findings carefully recorded and the registration of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer obtained at intervals according to any change in the other clinical signs.

As long as the pulse- and respiration-rates do not descend to 60 and to 16 and lower, respectively, and at the same time the ophthalmoscope does not reveal an edematous blurring of more than the nasal margins of the optic disks, or at most the nasal halves and temporal margins, and the spinal mercurial manometer registers the pressure of the cerebrospinal fluid as not being over 16 mm., and there are no localizing signs of convulsive seizures or paralyses of the extremities—then naturally no cranial operation should be considered advisable (unless there is a depressed fracture of the vault, which should always be elevated or removed); the expectant palliative treatment should be continued and if the signs of an increasing intracranial pressure do not appear, then the medical treatment alone is sufficient to obtain the best result possible: the operation of cranial decompression and drainage upon this group of patients—forming about two-thirds of the patients injured—is not only *not* justified but is *not* indicated, since these patients can "take care of" this mild increase of intracranial pressure of free hemorrhage and cerebral edema by the natural means of absorption alone, and therefore these patients should not be subjected to the risk of a cranial operation. If, however, the signs of an increasing intracranial pressure appear in the gradual descent of the pulse- and respiration-rates to 60 and to 16 and lower, respectively, the increase of the blood-pressure to 140 and higher, and, most important, the definite signs of an edematous obscuration of the optic disks to the degree of an early papilledema or even a measurable swelling of two diopters and higher (although this latter finding of "choked disks" is uncommon), and the pressure of the cerebrospinal fluid at lumbar puncture should now exceed 16 mm. and even 20 mm., and especially in the presence of an increasing paralysis of the extremities and epileptiform seizures of Jacksonian character or not, then there should be no hesitation whatever in advising an immediate operation of cranial decompression and drainage. To postpone the operation now until the more distinct signs of medullary compression occur, as would be indicated by the very slow and irregular Cheyne-Stokes type of pulse- and respiration-rates, with or without an increasing and profound unconsciousness, and a papilledema to the degree of even "choked disks," and a pressure of the cerebrospinal fluid of 24 mm. and higher—this condition would be a most dangerous one for the patient to reach; the early onset of the signs of medullary edema and collapse (a rapidly increasing pulse- and respiration-rate to 100 and 30 and higher, respectively) might appear within several hours and even earlier, and thus the certain death of the patient. Besides, the value of the operation of cranial decompression and drainage for these patients after they have entered the clinical stage of acute medullary compression is a doubtful one in that the mortality is high even with the immediate operative relief of the high intracranial pressure—that is, the medullary compression merges into the condition of medullary collapse before the beneficial effects of the cranial decompression are possible because the resistance of the cardiac and respiratory centres in the medulla has been so diminished by the high unrelieved intracranial pressure that an edema of the medulla occurs and once it occurs these patients all die—operation or no operation.

F ie only methods of estimating, in a crude way, the presence
cranial pressure were the descent and ratio of the pulse- and
s, associated with the height of the general blood-pressure—a

most unsatisfactory and late means of establishing definitely the necessity of a cranial operation; naturally, the mortality was therefore high, not only from an inefficient technic but chiefly from the fact that the operation was performed at such a late stage of acute medullary compression that the condition of medullary edema was imminent and at times even hastened by the operation itself.

The more accurate methods of ascertaining the intracranial pressure by means of the ophthalmoscope and the spinal mercurial manometer in addition to the pulse- and respiration-rates, blood-pressure and the presence or not of profound unconsciousness, paralyses and convulsions—this advance in the diagnostic methods has made it possible to anticipate these cruder clinical signs of an approaching medullary compression, so that the intracranial pressure can be relieved earlier, with greater safety and with a much lower mortality rate. It is not to be doubted that there are patients who have been in this stage of acute medullary compression with a pulse-rate of 50 and even lower, and yet they have recovered life without a cranial operation; their constitutional resistance to this high intracranial pressure has enabled them to withstand it successfully, and yet these patients (and I have followed a number of them) rarely if ever regain their former good health but are permanently damaged to a greater or less degree from having endured this high pressure over a period of days, and usually longer. It is, therefore, not only the immediate recovery of life of the patient which must be considered but also his future ultimate condition of normality, and there is no question that the early operation of cranial decompression and drainage affords these patients the best chance of complete recovery.

It is now realized, therefore, that the two periods in which no cranial operation should be performed upon these patients is, first, during the initial period of severe shock with a pulse-rate above 110, and then, second, the terminal period of medullary edema and collapse when the pulse- and respiration-rates have reached their lowest levels of 50 and 12 and even lower, respectively, and have begun to rise rapidly to 100 and 30 and higher, respectively. If a patient survives an operation during this first period of severe shock, then he recovers *in spite of* the operation, whereas if a patient should survive a cranial operation after the clinical signs of an acute medullary edema have appeared—well, it does not happen, and I have yet to see a patient recover from this extreme terminal condition—operation or no operation. In conclusion, as stated before, only about one-third of the patients having brain injuries require a cranial operation, while the other two-thirds can and do make excellent recoveries under the expectant palliative method of treatment. If the intracranial pressure, however, exceeds a height beyond which it is not considered probable that the expectant palliative method will be sufficient to obtain the best result as to life and future normality, then the ideal time for the cranial operation of decompression and drainage is before the period of acute medullary compression, and this can be ascertained by the more recent methods of examination—particularly the intelligent use of the ophthalmoscope and of the spinal mercurial manometer. If the patient is already in the stage of acute medullary compression, of lowered and irregular pulse- and respiration-rates, then the cranial operation at least offers the patient a definite chance to recover life, but the relief of the pressure afforded at that late period may not be sufficient to obtain the best result; it should, however, be attempted.

Choice of Operation.—In depressed fractures of the vault of the skull, the depressed area of bone should naturally be elevated or removed; if, however, there is present a marked increase of the intracranial pressure—at least above 16 mm.—then a subtemporal decompression should first be performed on the same side of the head as the depressed area of bone, and then the latter can be safely elevated or removed—at the same operation or at a second operation several days later. It is distinctly dangerous to elevate or remove a depressed area of the vault if the underlying dura is torn and the intracranial pressure is high, for fear that the adjacent cerebral cortex will be forced upward through the bony opening and thus be irreparably damaged. This unfortunate complication frequently occurs in these patients having a high intracranial pressure unless the precaution is first taken of relieving the pressure by means of a subtemporal decompression.

The osteoplastic "flap" operation is rarely of application and of value in these patients having brain injuries. Not only is it a much more formidable operation than the subtemporal decompression in that it is a greater shock to the patient by requiring a longer time—at least one hour and more, and the loss of blood is greater, but the intracranial lesion in these patients is rarely of such a situation and character that it can be definitely stated that the intracranial hemorrhage is limited to one particular portion of the brain, and therefore indicating that a certain cortical area must be exposed. In almost all of these patients, there are no accurate localizing signs pointing to more than a lesion of either cerebral hemisphere, and this results from the fact that the usual lesion is a subdural free hemorrhage, and supracortical rather than localized in the cerebral cortex itself, and also to the great frequency of an acute cerebral edema alone—that is, a "wet" edematous condition of the brain resulting from an excess of the cerebrospinal fluid; these conditions naturally do not present any definite localizing signs clinically other than the involvement of one cerebral hemisphere more than the other. Besides, to perform an osteoplastic "flap" operation over the more developed areas of the cerebral cortex and particularly above and adjacent to either motor tract and in the presence of a high intracranial pressure—the risk of a permanent damage to these important areas would be very great indeed; such impairment following these operations in the past almost discredited the operative treatment of selected cases of brain injuries—so much so that the patient was considered to have an equal chance of ultimate recovery without a cranial operation as with one—and possibly more so.

The advantage of the subtemporal route over other methods of cranial decompression is chiefly due to its anatomical relations; not only is the squamous bone underlying the temporal muscle the thinnest part of the vault of the skull and therefore less difficult to remove, but it exposes a part of the cortex most frequently involved in cases of brain injury with and without a fracture of the skull where the middle meningeal artery is torn or the temporo-sphenoidal lobe is lacerated, and in cases of abscess of the temporo-sphenoidal lobe following its usual cause—an otitis media; with little difficulty, the lower portion of the motor tract may be explored as well as the posterior portion of the frontal lobe; and on the left side the motor speech area is easily observed. Another important advantage is the fact that the part of the brain lying directly beneath the decompression opening is the cortex of the temporo-sphenoidal lobe—a comparatively silent area of the brain; for this reason any possible operative damage is not revealed clinically, and in patients having a high intracranial pressure, the protrusion of this part of the brain into the decompression opening does not produce paralyses, etc.,—a frightful result of "decompressions" at times performed over the parietal bones. That is, a subtemporal decompression relieves increased intracranial pressure without the danger of cerebral impairment; besides, it affords excellent drainage for the middle fossa of the skull at its lowest point—a very important consideration in patients having brain injuries.

Again, the thick overlying temporal muscle not only makes possible a firm closure but also allows the underlying bone to be removed so that a permanent decompression results with no danger of a hernia cerebri. The scalp is not weakened by drainage through the lower angle of the split temporal muscle and no unsightly protrusion occurs; the scar is always inside the hair-line. Besides, in men, the rim of the derby or straw hat affords some protection to the area of decompression although no protection is really necessary, as the temporal muscle is thick and thus the underlying cortex is more protected than the eyeball; besides, the underlying cortex itself is comparatively a silent area of the brain so that even if it were injured by some sharp object being thrust into the opening, no clinical signs would appear unless an acute infection should result; this possible remote complication has not occurred in any of the operated patients.

The vertical incision in this operation is far superior to the older method of a curvilinear incision over or at the parietal crest. Not only may the manual pressure-traction method of hemostasis be used much more effectively with the vertical incision, but the temporal artery is clamped at its lowest point at the very beginning of the operation and before the artery branches into numerous smaller vessels, whereas, in the curved incision, the many branches of the temporal artery are severed individually and each one must be clamped separately; again, it is easier to enlarge the bony opening downward to the base of the skull when the vertical incision is used—a very important point for drainage in cases of brain injury with and without a fracture of the skull. To preserve the strong attachment of the temporal muscle to the parietal crest is very difficult and even impossible when the usual curved incision is used; in this manner, the decompression may so weaken the side of the head that a hernia cerebri appears as the intracranial pressure increases; especially is this true in irremovable tumors of the brain. This complication is a most rare occurrence following a decompression performed with the vertical incision and with a careful regard for the attachment of the temporal muscle.

CHAPTER VIII

THE TECHNIC OF THE OPERATION OF SUBTEMPORAL DECOMPRESSION

THE usual general preparation of the patient for an operation is the restriction of diet and the administration of a soapsuds enema several hours before the operation. The entire head or merely the side of the head selected for operation is carefully shaved, either on the preceding night and a green soap poultice applied or in emergency patients the operative area is closely shaved just before the operation. Unless there are clinical signs indicating a lesion of the left cerebral hemisphere, the decompression to lower an increased intracranial pressure is always performed on the right side in order to avoid the motor speech area, which is situated in right-handed persons in

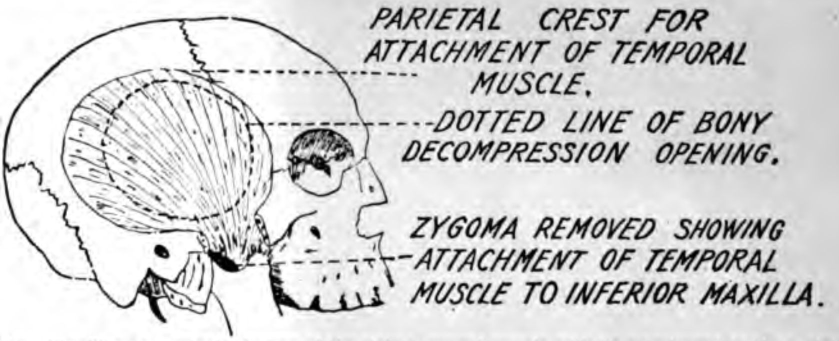
PARIETAL CREST FOR ATTACHMENT OF TEMPORAL MUSCLE.
DOTTED LINE OF BONY DECOMPRESSION OPENING.
ZYGOMA REMOVED SHOWING ATTACHMENT OF TEMPORAL MUSCLE TO INFERIOR MAXILLA.

FIG. 17.—The anatomical relations of the right temporal muscle with its attachment to the parietal crest above and to the inferior maxilla beneath the zygoma below; its diameter is usually three inches and more in adults, and forms an ideal protection for the underlying bony opening of the subtemporal decompression (indicated by the dotted line). The direction of the muscle fibres illustrates how easy it is to separate them longitudinally and vertically.

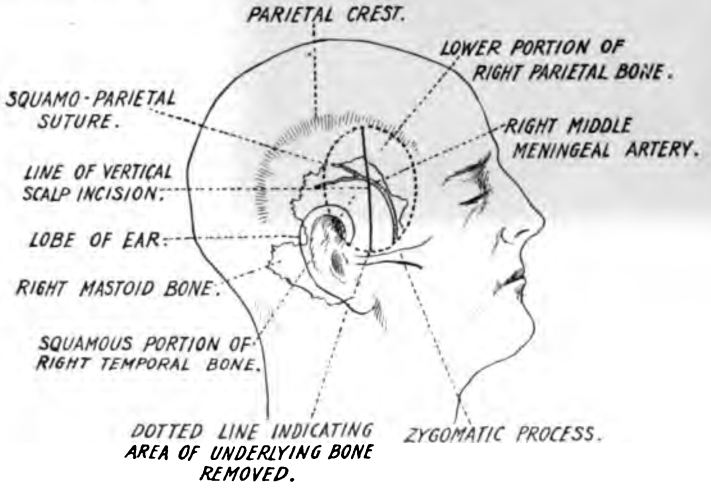
PARIETAL CREST.
LOWER PORTION OF RIGHT PARIETAL BONE.
SQUAMO-PARIETAL SUTURE.
RIGHT MIDDLE MENINGEAL ARTERY.
LINE OF VERTICAL SCALP INCISION.
LOBE OF EAR.
RIGHT MASTOID BONE.
SQUAMOUS PORTION OF RIGHT TEMPORAL BONE.
DOTTED LINE INDICATING AREA OF UNDERLYING BONE REMOVED.
ZYGOMATIC PROCESS.

FIG. 18.—The extent of the vertical scalp incision in relation to the underlying squamous portion of the right temporal bone and the lower part of the right parietal bone; the right middle meningeal artery is seen curving upward and backward. The dotted line of removed bone reaches the base of the skull—the middle fossa.

the posterior portion of the third left frontal convolution, and *vice vers* left-handed patients. The anatomical relations are illustrated in Figs. and 18. The patient is placed upon his back with the right shoulder sligl elevated by a sand-bag so that the right side of the head can be more ea made parallel to the horizontal plane of the table; in this manner, the op tive site is well exposed and it does not compel the operator, who stand the head of the table, and his assistants to assume tiring positions. anesthetist is seated under a sterile sheet at the waist of the patient, an this way he is entirely excluded from the field of operation. The anesthesi these patients requires the most skilful administration; especially is this t to avoid an extreme cyanosis and congestion during the induction of narcosis and also after the dura has been incised and the cerebral cortex exposed; coughing or even labored respiration at this stage of the operation may result disastrously by forcing the cortex through the bony opening so that even the cortex may be ruptured and serious hemorrhage occur. Dr. Charles S. Hunt, who has administered the anesthesia to most of my patients, uses a mixture of ether and oxygen very successfully (Fig. 19); he has found it necessary to deepen the narcosis just before the dura is incised, otherwise the sudden lowering of the intracranial pressure will allow the patient to show sign consciousness, coughing, etc.,—a complication to be feared greatly at stage of the operation.

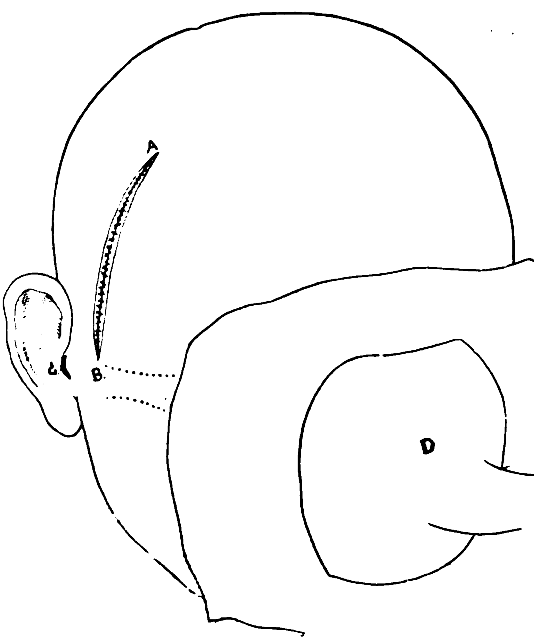

FIG. 19.—The superficial scalp incision in relation to the pa crest (*A*); and to the zygoma (*B*), and the external audito meatus (*C*). The ether-oxygen apparatus in position (*D*).

The side of the head and face are now carefully "scrubbed" with g soap and water for five minutes, and then alcohol (70 per cent.) is spon over the operative area. Iodine is only used in emergency cases when scalp cannot be thoroughly prepared; it tends to irritate the skin of m patients, especially children, and thus renders a secondary infection p ble. A superficial vertical incision of the skin (Fig. 20) is now mad indicate the extent of the scalp incision and then dry sterile towels clipped to the scalp at each side of this incision by towel clips (Figs. and 22); in this manner, the head is completely covered and th cannot become disarranged, so that there is littl

By using the method of bimanual pressure-traction at each side of the incision and the forefinger of the assistant firmly applied so as to compress the temporal artery as it ascends above the zygoma (Fig. 23), the incision can be made with very little loss of blood—a most important factor in all cranial operations; a cranial tourniquet cannot be used in this operation and the other methods of controlling hemorrhage of the scalp such as suturing the scalp, clipping of the scalp, etc., are not only time-consuming, troublesome, and even dangerous by increasing the risk of infection, but they are ineffective in many patients.

The incision is made vertically upward through the scalp from a point just above the zygoma and one-half inch anterior to the external auditory meatus, to the middle of the parietal crest and thus overlying the origin of the temporal muscle; it is about three to three and one-half inches in length, and is parallel to the fibres of the underlying temporal muscle. Small curved hemostats (Fig. 24) applied to the subcutaneous fascia are used to compress the branches of the temporal artery and then the temporal fascia is incised vertically (Fig. 25) and the fibres of the temporal muscle are split longitudinally and retracted, exposing the squamous portion of the temporal bone (Fig. 26). A sharp periosteal elevator is used to separate the muscle from the underlying bone; great care should be taken not to destroy the attachment of the muscle and its fascia to the parietal crest; otherwise, the closure of the temporal muscle will be greatly weakened.

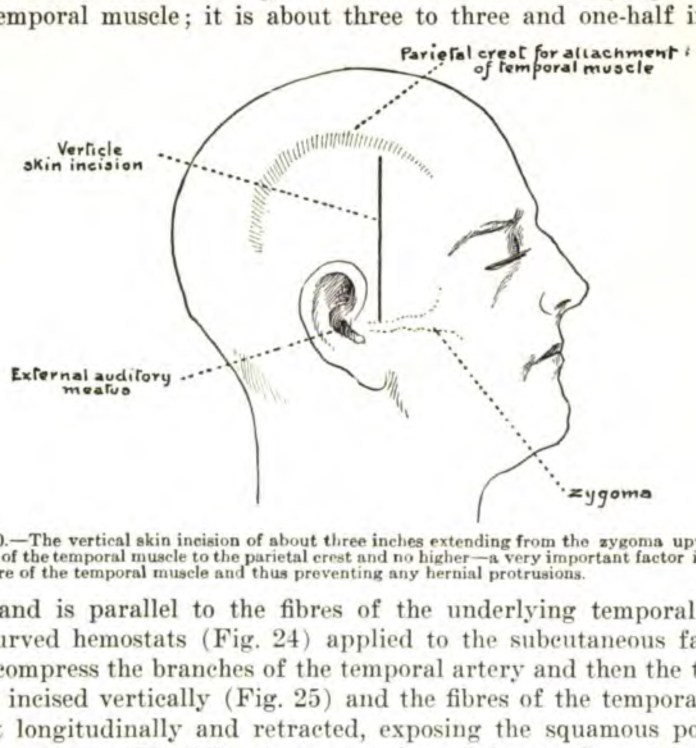

FIG. 20.—The vertical skin incision of about three inches extending from the zygoma upward to the attachment of the temporal muscle to the parietal crest and no higher—a very important factor in obtaining a firm closure of the temporal muscle and thus preventing any hernial protrusions.

The Doyen perforator and burr (Figs. 27 and 28) are now employed to make a small bony opening at the lower angle of the operative area, that is, the thinnest portion of the squamous bone (Figs. 29 and 30). Small rongeurs (Fig. 31) enlarge the opening (Fig. 32) until it is possible to use a larger rongeur having one blade levelled and flattened (Figs. 33 and 34), so that it can be easily and safely inserted between the dura and bone: frequent explorations and removal of adhesions between the dura and bone

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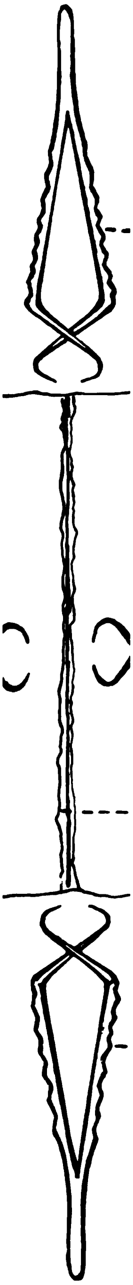

f covering the
skin itself; a
ear cannot be

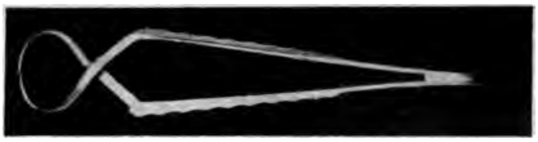

FIG. 22.—Top view (actual size) of a towel-clip which holds the towels firmly clipped to the scalp, thereby covering the skin and excluding it from the operative field and also preventing any slipping of the towels and consequently the great danger of infection.

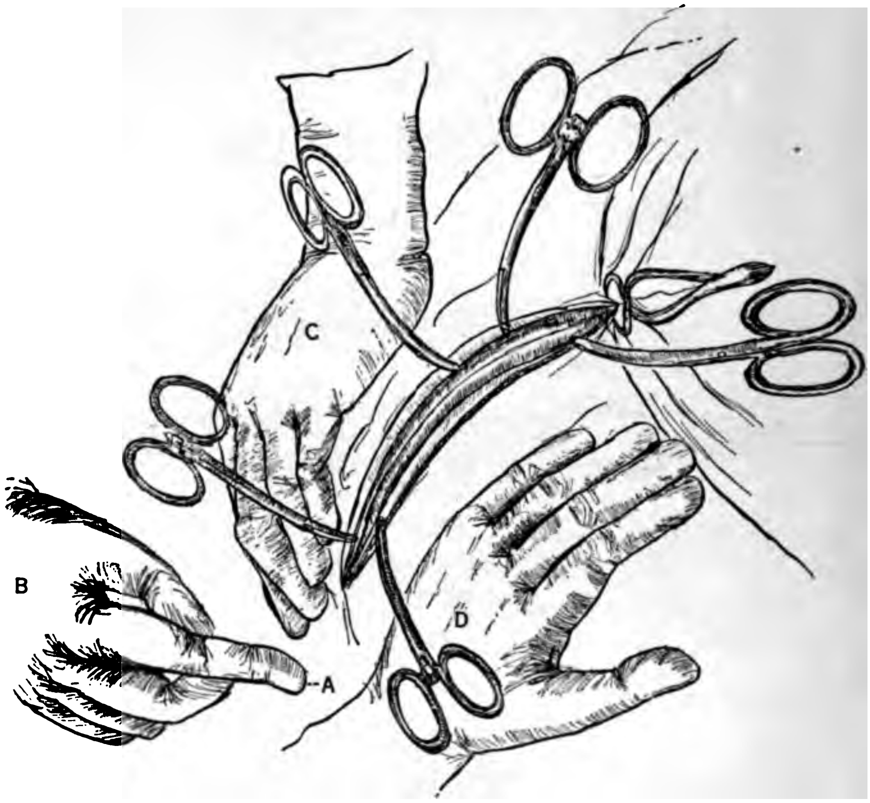

FIG. 23.—The use of bimanual pressure-traction at the side of the vertical incision of the scalp, and the compression of the temporal artery (*A*) by means of the forefinger of the assistant's right hand (*B*); with the left hand (*C*), the assistant compresses the vessels of the scalp on one side, while the left hand (*D*) of the operator is applied to the other side of the scalp incision. No other method of hemostasis, such as the use of a cranial tourniquet or a continuous scalp suture, is necessary.

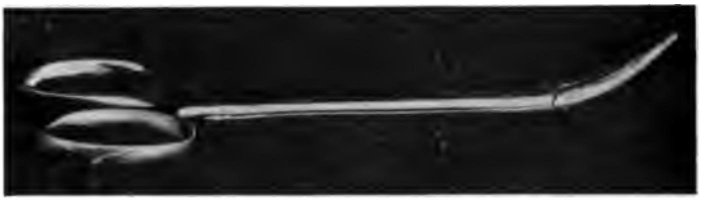

FIG. 24.—Lateral view (actual size) of the small curved hemostats used for clamping the subcutaneous fascia of the scalp and folding it backward, so as to compress the small vessels of the scalp and thereby avoid the necessity of clamping each vessel separately.

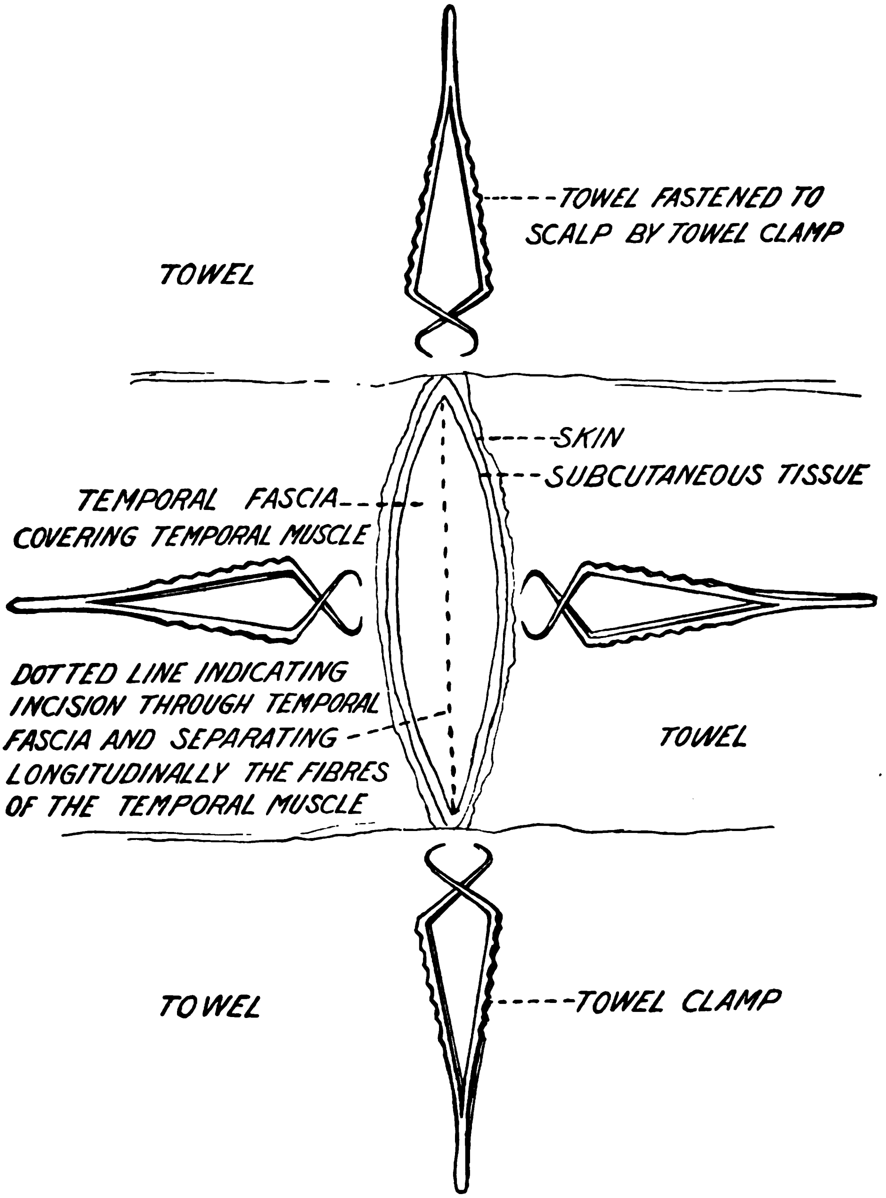
TOWEL FASTENED TO SCALP BY TOWEL CLAMP
TOWEL
SKIN
SUBCUTANEOUS TISSUE
TEMPORAL FASCIA COVERING TEMPORAL MUSCLE
DOTTED LINE INDICATING INCISION THROUGH TEMPORAL FASCIA AND SEPARATING LONGITUDINALLY THE FIBRES OF THE TEMPORAL MUSCLE
TOWEL
TOWEL
TOWEL CLAMP

FIG. 25.—The skin incision down to the underlying temporal fascia; by the method of bimanual pressure-traction upon the edges of the scalp incision, the bleeding is practically nil; small curved hemostats are now attached to the subcutaneous fascia so that any possible bleeding points are thus compressed by the hemostats being turned down.

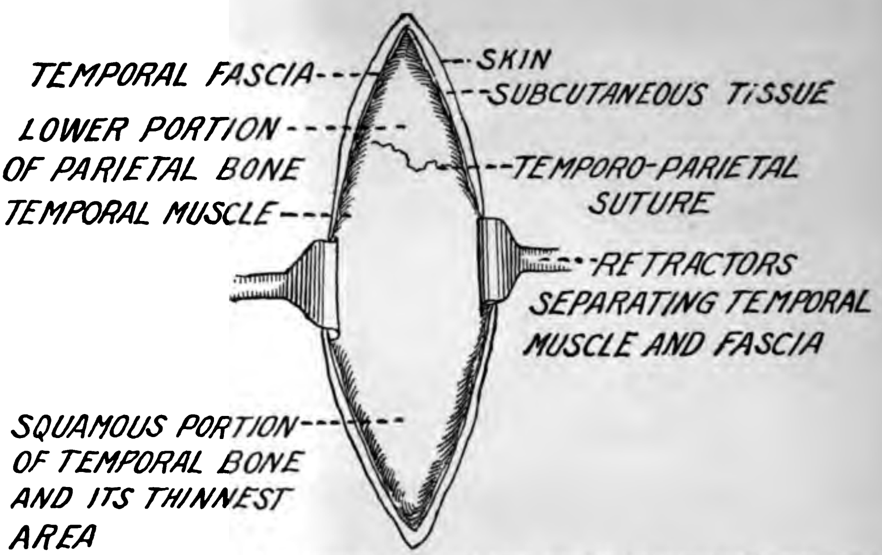
TEMPORAL FASCIA
LOWER PORTION OF PARIETAL BONE
TEMPORAL MUSCLE
SKIN
SUBCUTANEOUS TISSUE
TEMPORO-PARIETAL SUTURE
RETRACTORS SEPARATING TEMPORAL MUSCLE AND FASCIA
SQUAMOUS PORTION OF TEMPORAL BONE AND ITS THINNEST AREA

FIG. 26.—The retraction of the temporal muscle and fascia exposing the underlying lower portion of the parietal bone and the squamous portion of the temporal bone; an area of bone of three inches in diameter can thus be exposed and removed.

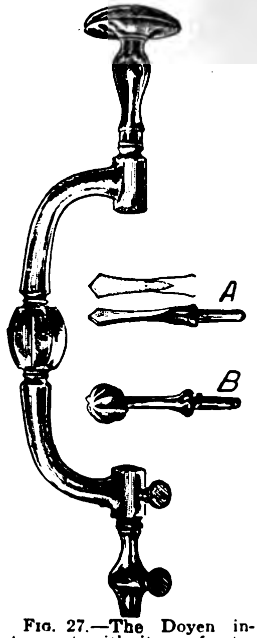

FIG. 27.—The Doyen instrument with its perforator (*A*) and its burr (*B*).

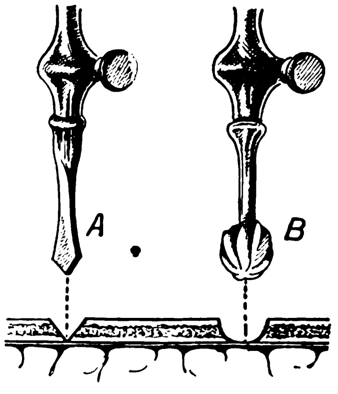

FIG. 28.—Diagrammatic cross-sections of the vault showing the safe method of opening the skull: a pin-point opening is made by the Doyen perforator (*A*) and then enlarged by the Doyen burr (*B*), so that the small rongeurs can be inserted; in this manner, the dura and the underlying cerebral cortex are never injured, and the middle meningeal artery can be avoided; the use of even a small trephine is thus contraindicated.

with the dural separator (Fig. 35) will prevent the dura from being torn. In this manner, a circular opening as large as possible under the temporal muscle is made, extending from the base of the skull up to the parietal crest and having a diameter of three to three and one-half inches.

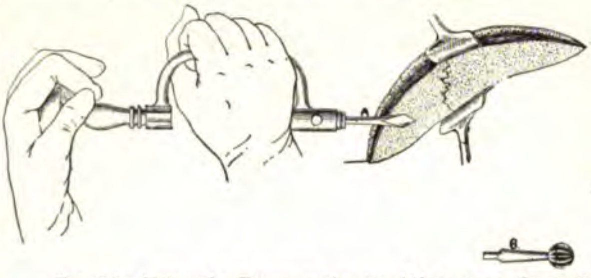

FIG. 29.—Using the Doyen perforator (*A*) to open the skull at the lower angle of the incision—the thinnest portion of the squamous bone. The Doyen burr (*B*) is next used to enlarge this opening for the insertion of the small rongeurs.

Before opening the dura, it is very important that all oozing from the bony margins should be stopped; the best method for controlling this bleeding from the diplöe and its sinuses is the rubbing of bone-wax into the edges of the bone and it is surprising how quickly this troublesome complication is overcome. Dr. Norman Sharpe has formulated a bone-wax which is most effective; its composition is as follows:

White Wax	7 parts
Almond Oil	2 parts
Salicylic Acid	1 part

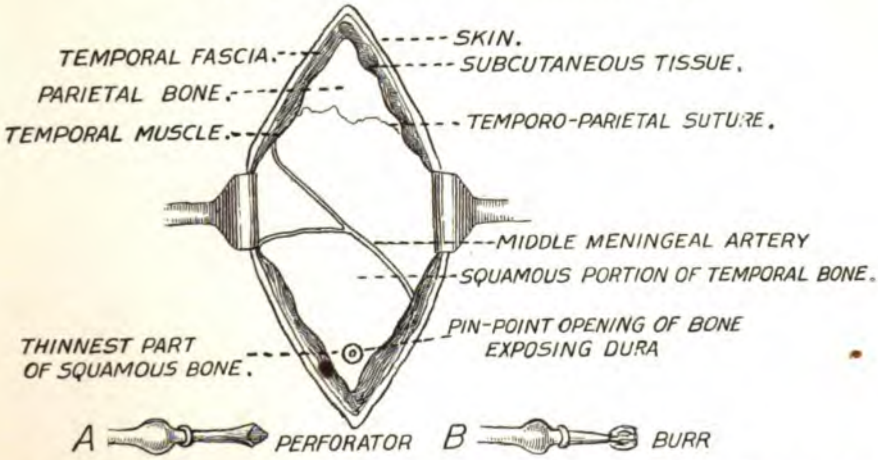

FIG. 30.—At the lowest and therefore the thinnest part of the squamous bone, the Doyen perforator (*A*) is used to make a pin-point opening down to the dura, and then the Doyen burr (*B*) enlarges the opening down to the dura so that small rongeurs can be inserted to remove the bone; care is taken to avoid an injury to the adjacent middle meningeal artery.

Keep in a 5 per cent. solution of carbolic acid. This wax may be sterilized before each operation, and then allowed to cool so that it hardens and is easily moulded; small pellets, the size of peas, are then rubbed into the oozing bone. It is a most effective method of plugging the middle meningeal artery when it channels the bone; it seems to me that it might be used in operations upon bone elsewhere, such as extensive resections of bone, etc.; it is far superior to the old method of using wooden pegs in cranial surgery.

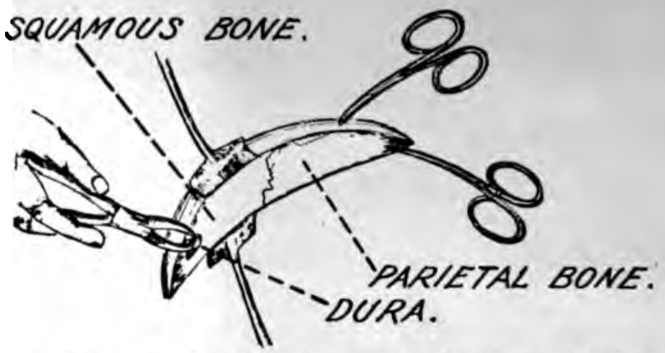

Fig. 31.—Small rongeurs enlarging the bony opening after the use of the Doyen burr.

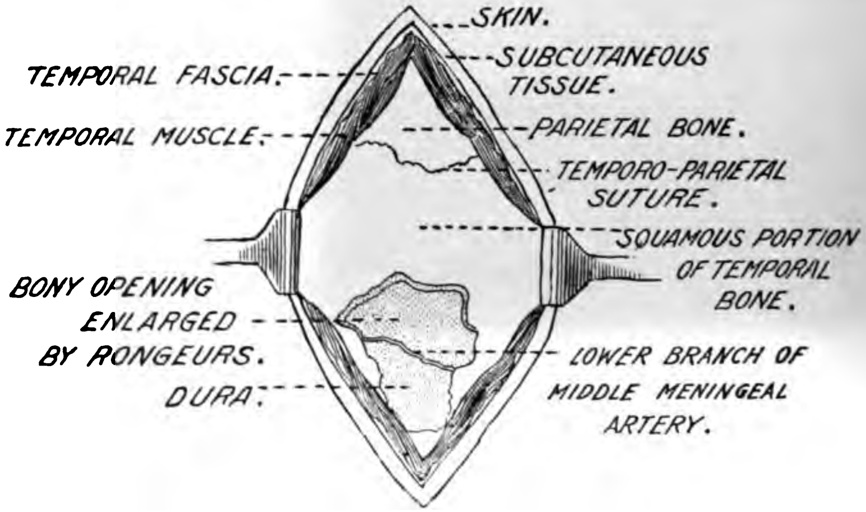
SKIN.
SUBCUTANEOUS TISSUE.
TEMPORAL FASCIA.
TEMPORAL MUSCLE.
PARIETAL BONE.
TEMPORO-PARIETAL SUTURE.
SQUAMOUS PORTION OF TEMPORAL BONE.
BONY OPENING ENLARGED BY RONGEURS.
DURA.
LOWER BRANCH OF MIDDLE MENINGEAL ARTERY.

Fig. 32.—Bony opening being enlarged by rongeurs backward, downward and upward, but not forward until last on account of the middle meningeal artery lying adjacent to the anterior portion of the bony exposure; if the artery should be torn, the hemorrhage can be very easily controlled either with bone wax, a silver clip, or, if necessary, by a small packing of gauze tape. The final size of the bony opening is about 3 inches in diameter.

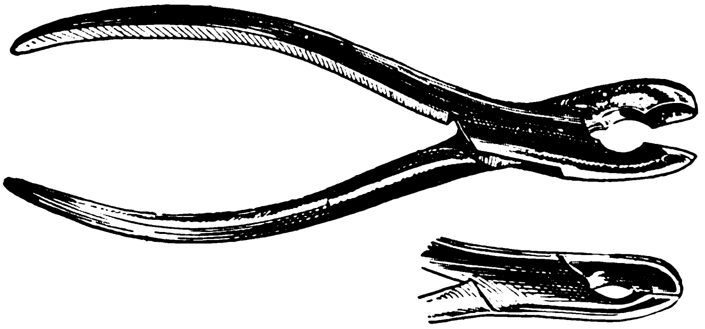

Fig. 33.—Larger bone rongeurs, having the thin lower blade bevelled for insertion between the dura and the overlying bone, which can now be safely removed at no risk to the dura itself.

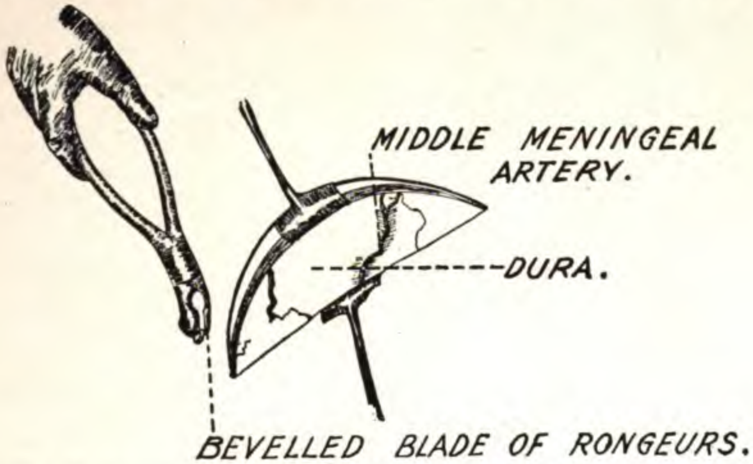

FIG. 34.—Bony opening being extended by larger rongeurs having one blade flattened and bevelled so that it can be easily inserted between the dura and the overlying bone without danger of tearing the dura itself.

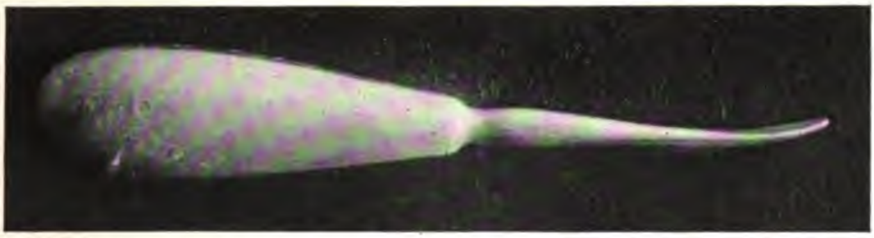

FIG. 35.—Lateral view (actual size) of the dural separator for insertion between the bone and the underlying dura and the removal of dural adhesions to the bone so that the danger of tearing the dura by the rongeurs is practically *nil*.

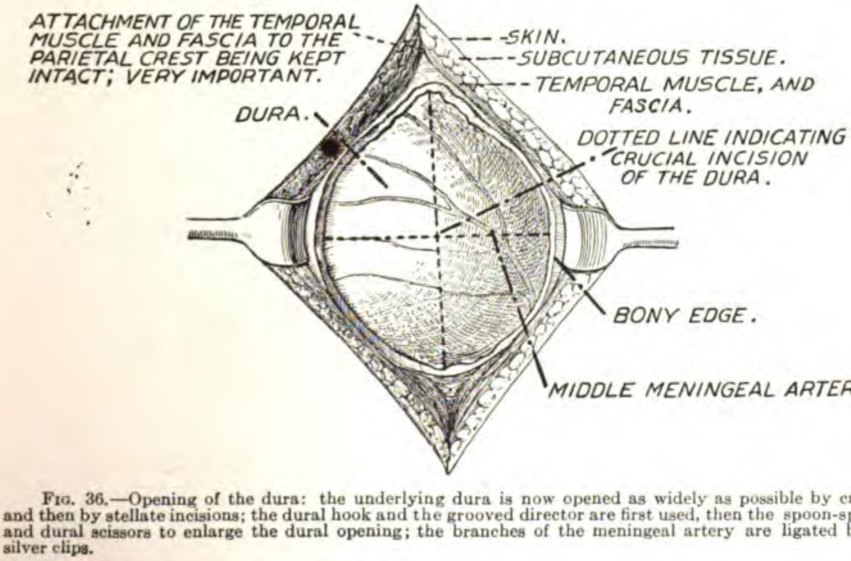
ATTACHMENT OF THE TEMPORAL MUSCLE AND FASCIA TO THE PARIETAL CREST BEING KEPT INTACT; VERY IMPORTANT.
SKIN.
SUBCUTANEOUS TISSUE.
TEMPORAL MUSCLE, AND FASCIA.
DURA.
DOTTED LINE INDICATING CRUCIAL INCISION OF THE DURA.
BONY EDGE.
MIDDLE MENINGEAL ARTERY.

FIG. 36.—Opening of the dura: the underlying dura is now opened as widely as possible by crucial and then by stellate incisions; the dural hook and the grooved director are first used, then the spoon-spatula and dural scissors to enlarge the dural opening; the branches of the meningeal artery are ligated by the silver clips.

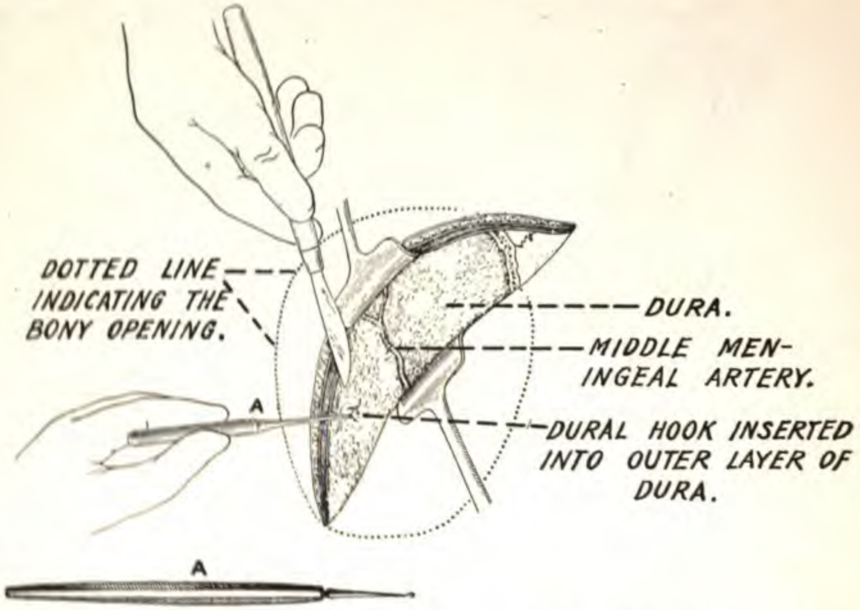
DOTTED LINE INDICATING THE BONY OPENING.
DURA.
MIDDLE MENINGEAL ARTERY.
A
DURAL HOOK INSERTED INTO OUTER LAYER OF DURA.
A

Fig. 37.—Small dural hook (A) inserted into the outer layer of the dura and being used to elevate the dura from the underlying cerebral cortex in order that a small dural incision may be safely made.

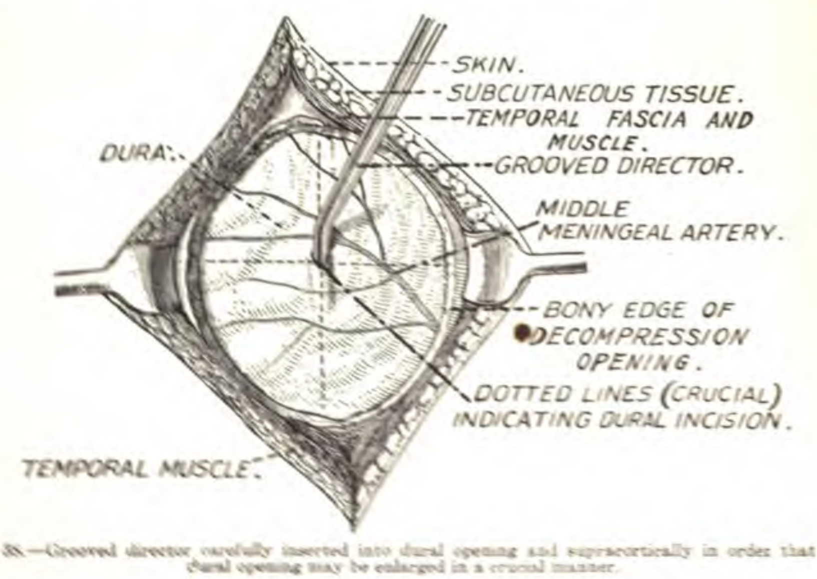
SKIN.
SUBCUTANEOUS TISSUE.
TEMPORAL FASCIA AND MUSCLE.
DURA.
GROOVED DIRECTOR.
MIDDLE MENINGEAL ARTERY.
BONY EDGE OF DECOMPRESSION OPENING.
DOTTED LINES (CRUCIAL) INDICATING DURAL INCISION.
TEMPORAL MUSCLE.

Fig. 38.—Grooved director carefully inserted into dural opening and supracortically in order that the dural opening may be enlarged in a crucial manner.

In fractures of the skull, the middle meningeal artery is frequently torn as it channels the bone of the vault, so that it is a very simple matter to remove the extradural clot and then plug the bony channel of the bleeding vessel with the wax.

The dura is now incised in a crucial manner (Fig. 36) by carefully cutting through its outer layer first with a sharp knife, and then elevating the dura from the underlying cortex by means of the small dural hook inserted into its outer layer (Fig. 37); the inner layer can then be safely incised until a small pin-point opening is made. A grooved director bent almost at right angles may now be carefully inserted and the dural opening enlarged by cutting the dura upon the director (Figs. 38 and 39). When the dural incision is one inch in length, it is easier and faster to insert a spoon-shaped spatula (Fig. 40) beneath the dura and then to cut the dura with a sharp pair of scissors; this method is not only safer, but it allows the dural incisions to approach the dural vessels as closely as possible so that these vessels may be clamped before being cut. Not only is it time-consuming and troublesome to ligate the dural vessels with silk or catgut, but it is dangerous to insert a needle beneath the vessels before the dura has been incised for fear of puncturing one of the many cortical vessels lying beneath, and thus complicating the operation very much indeed; if the decompression is to be performed, it should at least not injure the brain. An excellent method of clamping the dural vessels is the application of small silver V-shaped clips to them and then the dura and its vessels may be safely cut between each pair of clips and no bleeding results (Figs. 41 and 42). These silver clips may be left upon the dural vessels, and I have never seen any ill effects occur; in five cases at autopsy within two years after operation, the clips were found *in situ* and no tissue reaction had occurred, so that they are apparently not irritating foreign bodies; in some patients when the dura was very vascular, as many as eight clips were applied, although the usual number is not more than three or four. The clips are made by wrapping German silver wire, No. 24, snugly around a rectangular rod and then bisecting the rolls (Fig. 43); V-shaped clips are thus formed

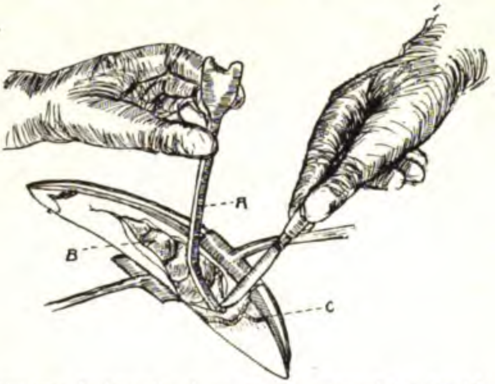

FIG. 39.—Incising the dura upon the grooved director (*A*) downward to the lower branch of the middle meningeal artery (*C*), which is ligated by two silver clips and then severed between them; the cerebral cortex (*B*) is thus exposed.

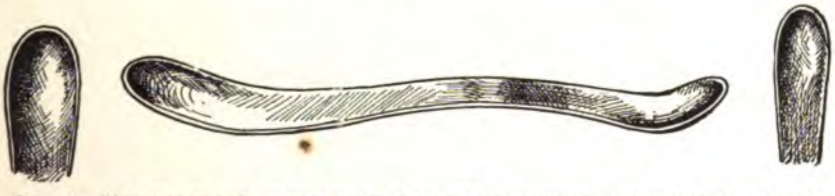

FIG. 40.—The spoon-spatula—a very useful instrument for protecting the underlying cerebral cortex when cutting the dura with the small dural scissors; it is also of much value in facilitating the exploration of the adjacent cerebral cortex beneath the bony margins of the decompression opening.

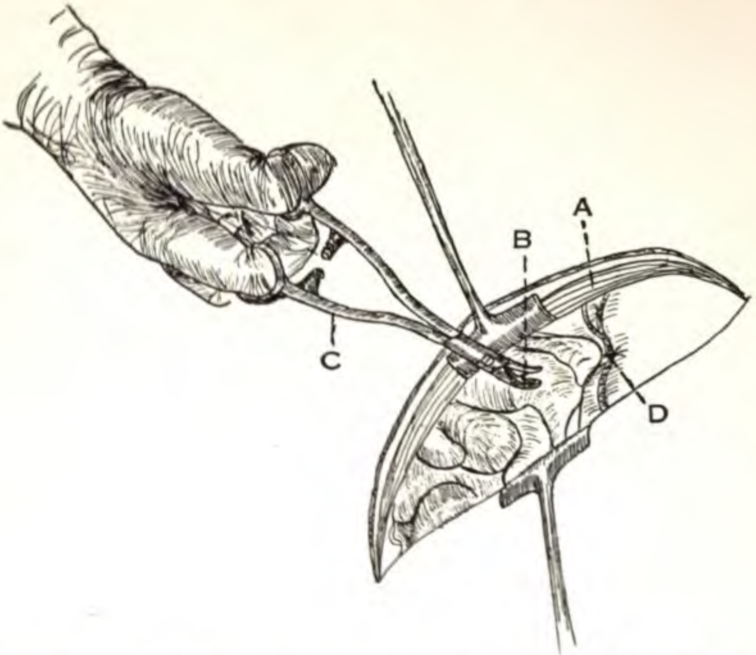

FIG. 41.—The ligation of the middle meningeal artery (*A*) in the dura by means of a silver clip (*B*) being compressed by the clip-holding forceps (*C*) about the vessels; one clip (*D*) has already been applied.

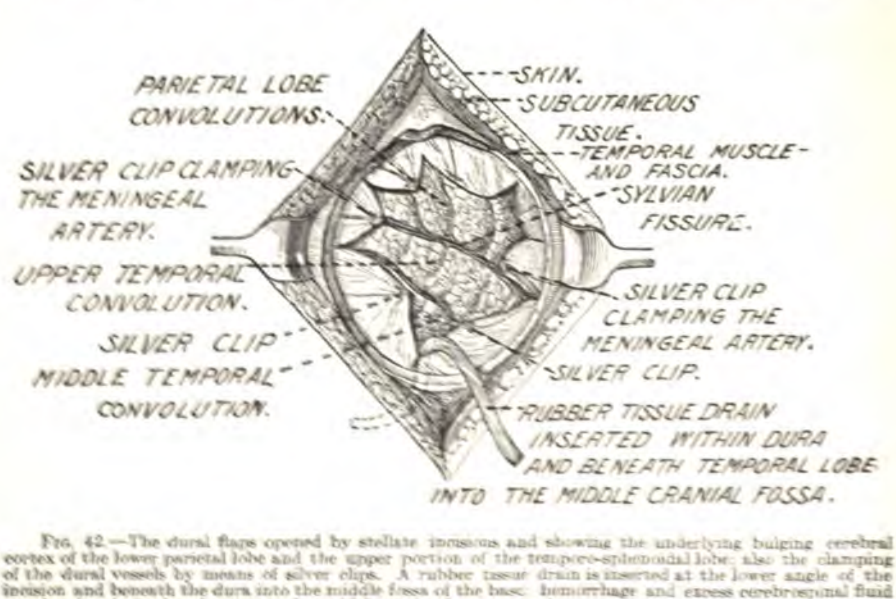
PARIETAL LOBE CONVOLUTIONS.
SKIN.
SUBCUTANEOUS TISSUE.
TEMPORAL MUSCLE AND FASCIA.
SILVER CLIP CLAMPING THE MENINGEAL ARTERY.
SYLVIAN FISSURE.
UPPER TEMPORAL CONVOLUTION.
SILVER CLIP CLAMPING THE MENINGEAL ARTERY.
SILVER CLIP
MIDDLE TEMPORAL CONVOLUTION.
SILVER CLIP.
RUBBER TISSUE DRAIN INSERTED WITHIN DURA AND BENEATH TEMPORAL LOBE INTO THE MIDDLE CRANIAL FOSSA.

FIG. 42.—The dural flaps opened by stellate incisions and showing the underlying bulging cerebral cortex of the lower parietal lobe and the upper portion of the temporo-sphenoidal lobe; also the clamping of the dural vessels by means of silver clips. A rubber tissue drain is inserted at the lower angle of the incision and beneath the dura into the middle fossa of the base; hemorrhage and excess cerebrospinal fluid can thus be drained and a "water-logged" brain be decompressed.

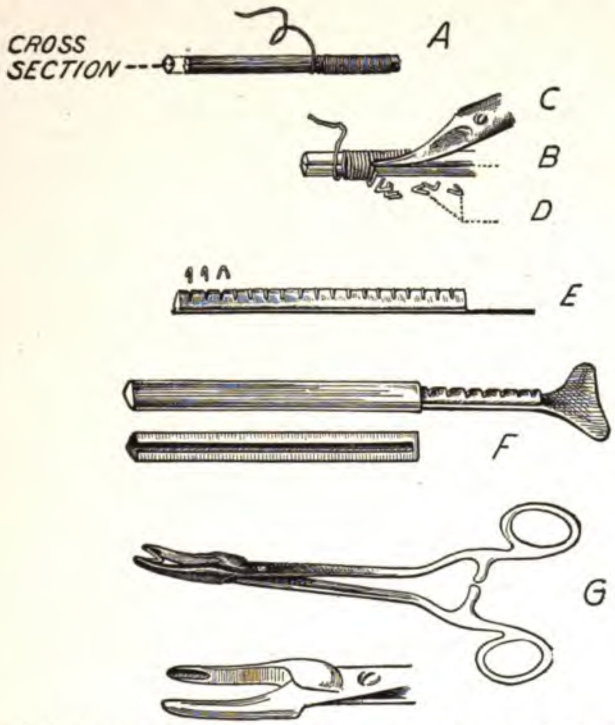

FIG. 43.—Method of making silver clips for the ligation of the dural vessels: silver wire (No. 24) is wrapped snugly around the rectangular grooved bar (*A*), and then cut in the groove on each side (*B*), by the wire-cutting scissors (*C*); the V-shaped silver clips (*D*) are placed in the grooves of the holder (*E*), inserted into the cover (*F*), sterilized and are then ready for use. The grooved forceps (*G*) for holding the silver clips facilitate their application to the dural vessels which are compressed by the closure of the forceps and the clips thus ligate the vessels by remaining *in situ*; the vessels may thus be cut safely and rapidly and no bleeding occurs; no ligatures are therefore necessary.

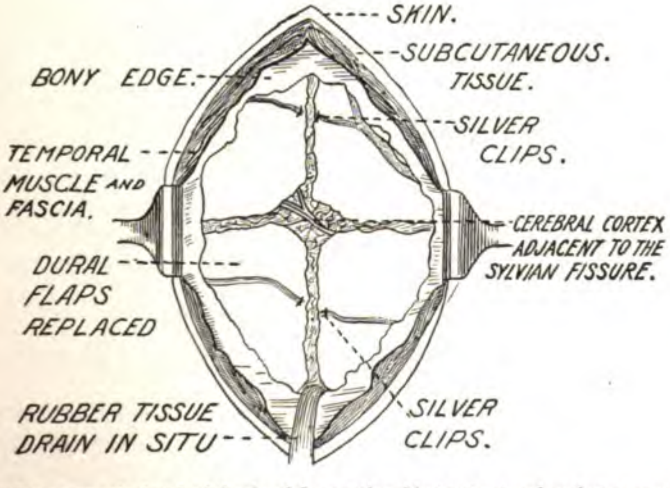
SKIN.
SUBCUTANEOUS. TISSUE.
BONY EDGE.
SILVER CLIPS.
TEMPORAL MUSCLE AND FASCIA.
CEREBRAL CORTEX ADJACENT TO THE SYLVIAN FISSURE.
DURAL FLAPS REPLACED
RUBBER TISSUE DRAIN IN SITU
SILVER CLIPS.

FIG. 44.—Diagrammatic sketch of the dural flaps replaced but not sutured so that a permanent decompression results; the silver clips can be seen clamping the dural branches of the middle meningeal artery; the rubber tissue drain is left *in situ*. Closure of the incision is now begun.

and after sterilization these can be put in a clip holder (similar to a hemostat with a grooved end) and clipped upon the dural vessel. This method saves much time and entails no risks.

The dural opening is thus enlarged in a crucial or stellate manner until the bony margins of the decompression are reached. It is very important to incise the dura downwards to the very base of the skull so that the middle fossa of the skull can be easily and freely drained—so essential in all brain injuries with and without a fracture of the skull and associated with an edematous, swollen brain with or without hemorrhage (Fig. 44). Through this opening any underlying pathological lesion can be dealt with freely and safely; large subdural clots may be removed in brain injuries, while small tumors can be removed and abscesses drained. Aided by the spoon-shaped spatula and a good electric headlight, the neighboring areas of the frontal lobe, the parietal lobe and the temporal lobe may be accurately explored for any cortical lesion. If the cerebral tension is very high, then the ipsolateral ventricle may be drained by the ventricle puncture needle; all parts of the temporo-sphenoidal lobe and even the posterior portion of the frontal lobe and the lower portion of the parietal lobe can be accurately explored in the same manner, as in cases of suspected brain tumor and abscess formation.

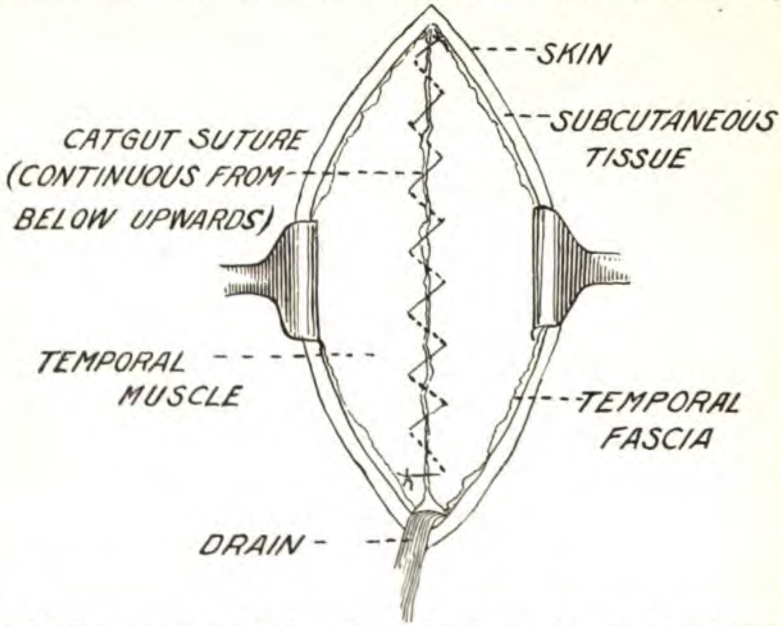

FIG. 45.—Diagrammatic sketch of the routine use of continuous catgut suture (from below upward) to approximate rapidly the fibres of the entire temporal muscle in one layer over the bony opening; if the intracranial pressure is extreme, then interrupted catgut or silk sutures in two layers may be advisable.

After the cerebral lesion has been removed or drained, or if merely the relief of intracranial pressure is desired, then a rubber tissue drain of one-quarter of an inch in width and several layers in thickness is inserted at the lower angle of the wound and inside the dura beneath the temporo-sphenoidal lobe as far as possible; in this manner, excellent drainage is afforded the middle cranial fossa; a second drain of rubber tissue is frequently inserted subdurally at the upper angle of the incision. Before the closure of the opening, it is important that there should not remain any bleeding points, no matter how small; small cotton pledgets wet in warm saline solution are frequently sufficient in many cases of cortical oozing, or small pieces of the temporal muscle applied to the bleeding point and then compressed for a few seconds will stop a most troublesome oozing. When tumors are removed, then packs of sterile cotton, either dry or wet in warm saline solution and pressed into the cavity of the enucleated mass, will quickly prevent a large hemorrhage; it is rarely necessary to let an intracranial packing remain *in situ*.

The drain having been inserted beneath the temporo-sphenoidal lobe, the temporal muscle is now sutured (Figs. 45, 46 and 47) with continuous plain catgut (Nos. I and II in children and adults, respectively); then the temporal fascia with interrupted catgut and black silk alternately (Figs. 48, 49, and 50), and finally the subcutaneous tissues with interrupted catgut (No. 1) (Fig. 51); the vessels of the scalp are not ligated as the mere suturing of the subcutaneous tissues is sufficient to compress their vessels; at times, the temporal artery is separately ligated. The skin is carefully approximated by interrupted sutures of fine black silk (Fig. 52). Dry gauze pads are now applied to the operative area, and after a cotton pad well covered with sterile vaseline is placed behind the lobe of the ear to prevent its being pressed against the skull and thus causing severe pain, the usual bandage of rolled gauze is applied and held in place by several strips of adhesive plaster (Fig. 53).

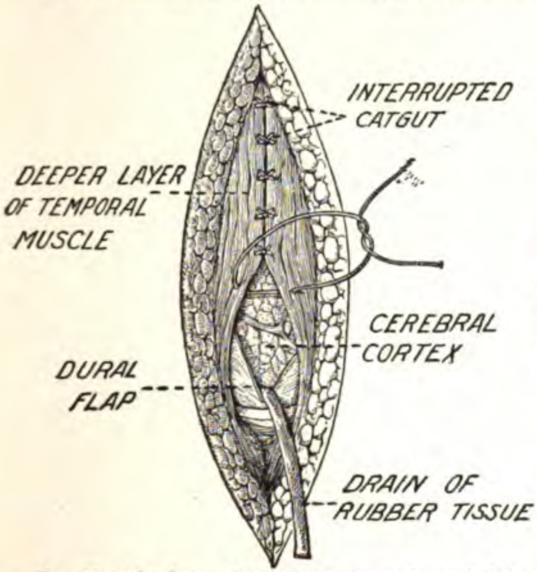

Fig. 46.—The deeper fibres of the temporal muscle being approximated by interrupted catgut or silk sutures (from above downward and in two layers) in patients when the increased intracranial pressure is extreme. The dura is never resutured, nor could it be when the intracranial pressure is high without danger of damage to the underlying cerebral cortex.

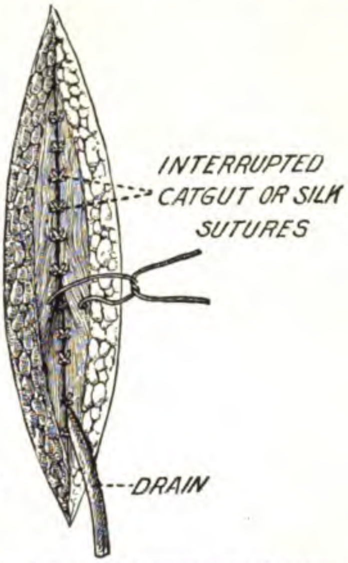

Fig. 47.—The upper fibres of the temporal muscle being sutured with interrupted catgut as the second layer of sutures.

In the operation of subtemporal decompression to lower an increased intracranial pressure, the dural opening is never sutured together; in the first place, if there is much intradural pressure, it would be impossible to approximate the edges of the dura on account of the cerebral protrusion,

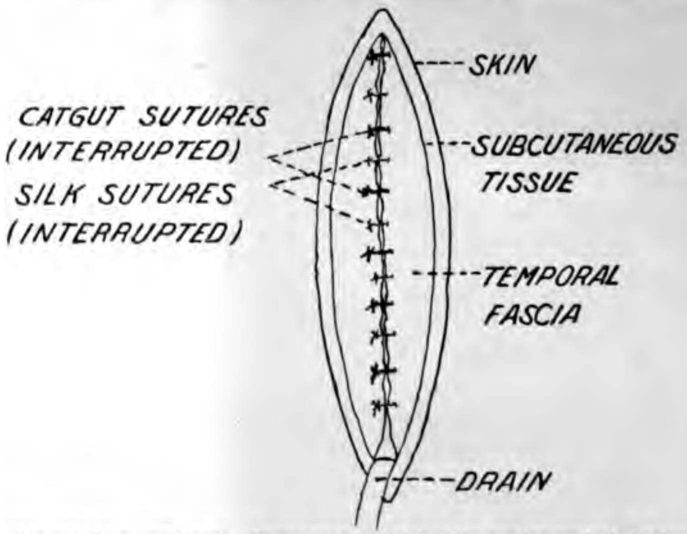

FIG. 48.—*Closure of temporal fascia.* Diagrammatic sketch of the routine method of using interrupted small black silk sutures, alternating with interrupted catgut sutures for the closure of the temporal fascia and thus making a firm approximation possible; the silk sutures are permanent sutures and prevent an opening of the incision if the catgut sutures should be absorbed too early.

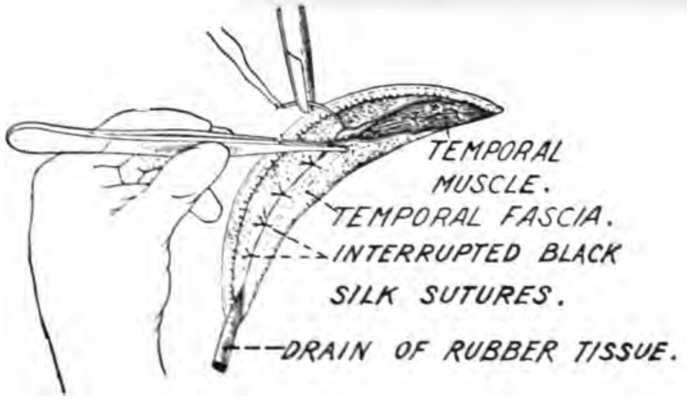

FIG. 49.—Inserting the interrupted black silk sutures for the approximation of the edges of the temporal fascia (from below upward).

and secondly, to resuture the dura would be to destroy the object of the decompression—the relief of intracranial pressure: for in adults, the dura is inelastic, so that there can be no real decompression if the dura is unopened or resutured after being opened. There is no danger apparently in letting the dura remain open: adhesions do not form, and as revealed in three cases at autopsy, a new dura was present. The overlying muscle forms a safe protecting covering.

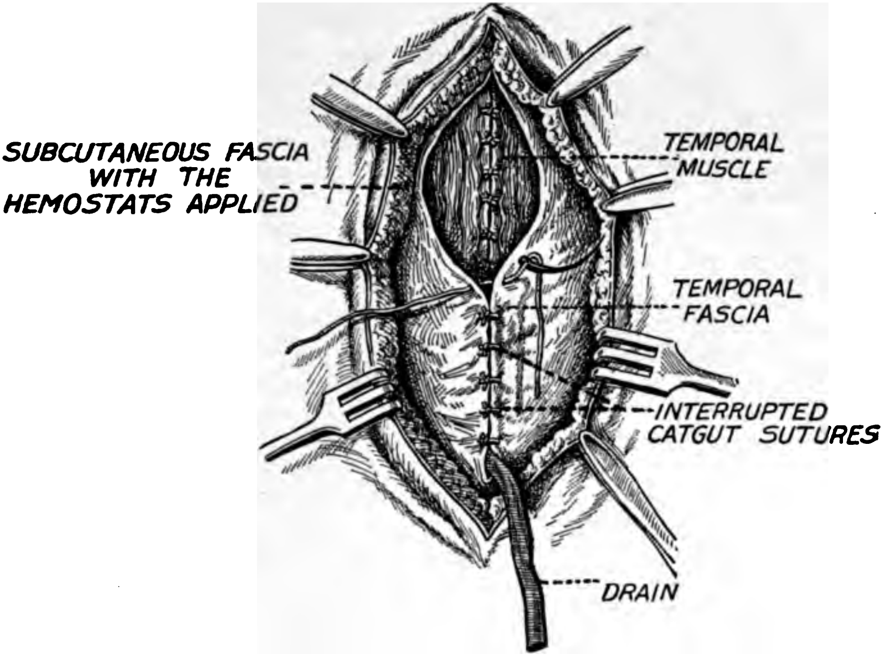
SUBCUTANEOUS FASCIA WITH THE HEMOSTATS APPLIED
TEMPORAL MUSCLE
TEMPORAL FASCIA
INTERRUPTED CATGUT SUTURES
DRAIN

Fig. 50.—Interrupted catgut sutures to alternate with the black silk sutures in the temporal fascia.

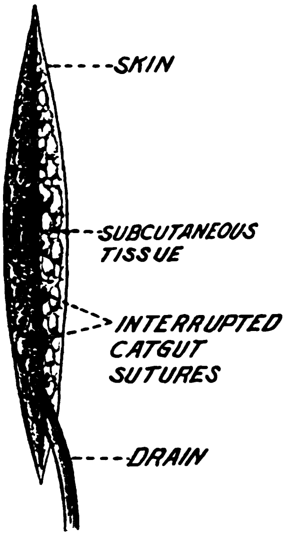

Fig. 51.—*Closure of the subcutaneous fascia.* It is sutured with interrupted catgut; continuous catgut (loosely) may be used in children in order to hasten the closure in emergency conditions.

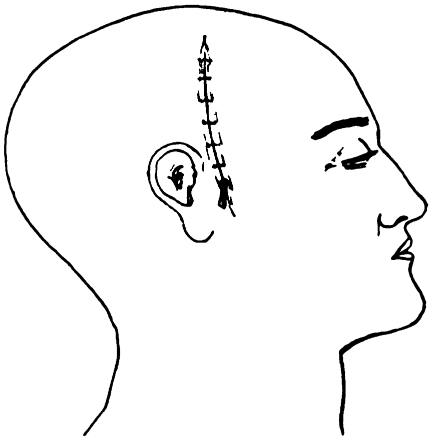

Fig. 52.—*Closure of the scalp.* Careful approximation of the skin incision with interrupted fine black silk sutures; the drain of rubber tissue is allowed to remain *in situ* for one or two days, according to the amount of drainage of blood and cerebrospinal fluid. Sterile gauze dressing and bandage are now applied.

Post-operative Treatment.—Beside the routine hospital treatment, the following post-operative measures are advisable:

I. Raise the head and shoulders with two or three pillows to an elevation of 25 to 30 degrees; if the shock of the intracranial injury and of the operation is severe, then allow the patient to remain "flat" in bed. If the temperature should descend rapidly to 104° and higher, and especially in children, and in the absence of pulmonary complications, it is then advisable to lower the head and trunk by elevating the foot of the bed and very frequently the temperature will soon descend to 102 and even lower.

II. Water by mouth as soon as the nausea ceases—both to replace the loss of blood and cerebrospinal fluid and to lessen the thirst and thereby the restlessness of many of these patients.

III. Rectal enemata of 400 c.c. of warm normal saline solution immediately, and repeated every four hours for at least 24 and even 36 hours. If the patient is in a severe condition of operative shock, then 400 c.c. of hot black coffee per rectum is administered immediately after the operation, and this is repeated two and then four hours later, if deemed necessary. Formerly, urotropin, gr. v–x, were given in the rectal saline every four hours; it was believed to inhibit the growth of bacteria in the cerebrospinal fluid, and in this way, a possible infection and meningitis were avoided; its real efficacy is doubtful since the cerebrospinal fluid is mildly alkaline and urotropin is effective in only acid media, such as the urine, etc.; it can, however, do no harm, and it may do some good.

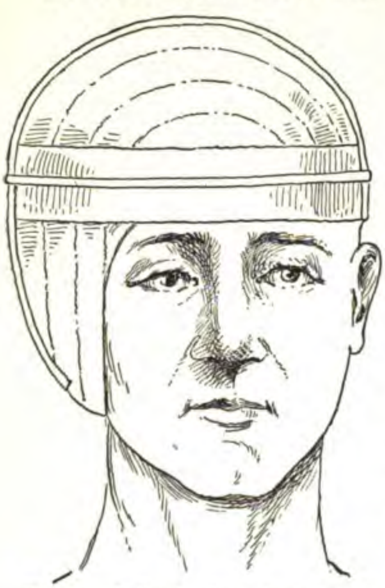

Fig. 53.—Roller bandage of sterile gauze applied to the head and firmly anchored by several adhesive strips. A small cotton pad placed behind the ear lobe prevents its painful compression against the head; the opposite ear is not covered.

IV. The temperature, pulse and respiration should be recorded each hour for twelve hours, and then every two hours for at least 36 hours; in this manner, the general condition of the patient is ascertained, a rare secondary hemorrhage or an increasing intracranial pressure affecting the medulla may be suspected early and the appropriate treatment immediately instituted; repeated ophthalmoscopic examinations are essential.

V. Morphia, gr. ⅛ hypodermically for restlessness, and repeated after one hour, if necessary.

The room should be cool and darkened, quietness being enforced. On the day following the operation, liquids, except meat juices and milk, may be given. On the second day post-operative, the first dressing is made; the drain is now removed, allowing clear or even slightly blood-tinged cerebrospinal fluid to trickle out; drainage of bloody cerebrospinal fluid and even blood itself in these patients during the first twenty-four hours after operation, may be profuse and sufficient to soak through the dressings. At least one-third of the skin sutures can now be removed. All liquids except meat juices may now be given; and, on the fourth day post-operative, soft diet. On the fifth day post-operative, the second dressing is made; all skin sutures are removed and the patient is placed upon a light diet. The average duration of the hospital residence is ten to fourteen days; no patient, however, should be permitted to return home unless his general condition is excellent and the operative area depressed or at least flush with the surrounding scalp (but not tense and bulging), and thus indicating that the increased intracranial pressure has been relieved; ophthalmoscopic examinations of the fundi are very helpful in determining the gradual subsidence of the edematous obscuration of the details of the optic disks.

Upon returning to their homes, these patients should not be permitted to lead active lives for a period of three to six months; even the least injured of them are not so stable emotionally for several months as before the injury, and therefore it is essential for them to avoid all excitement and especially that of a strenuous business life; their social and domestic activities should be restricted to a minimum. These patients should be examined at frequent intervals for a year and even longer in order that no untoward signs should be overlooked. Their diet should be a light one with no meat, meat-soup, tea or coffee for at least six months; and most important of all, no alcohol in any form whatsoever—the danger of convulsive seizures is an ever-present one on account of the increased cortical irritability of the brain and the after-treatment must be directed to its lessening; many of the future complications possible in these patients result from the neglect of these precautions. A careful regulation of the bowels so that a daily movement occurs is also of much importance. All severe mental and emotional strains should be avoided for at least a period of one year and if possible for several years.

CHAPTER IX

OBSERVATIONS REGARDING THE OPERATION OF CRANIAL DECOMPRESSION

THE field of neurological surgery has so broadened during the past fifteen years as the result of the pioneer work of Horsley, von Eiselsberg, and Krause, and in this country of Cushing, that a number of neurological conditions that were formerly considered hopeless are now amenable to improvement at least, and in some early cases even a cure may be expected. This advance has been due chiefly to earlier diagnosis, an improved surgical technic and surgical judgment, and to better team-work of the surgeon and the neurologist.

Earlier diagnosis of many intracranial conditions is now possible mainly as the result of the more general and intelligent use of the ophthalmoscope; it is now commonly recognized and appreciated that the condition of marked papilledema and "choked disks" is the end-result of preëxisting pressure signs observable in the fundus of the eye; no longer is it necessary to wait until a measurable papilledema occurs before it can be definitely stated that an increase of the intracranial pressure is present. Beside the early fundal signs ascertainable by an ophthalmoscopic examination, the most accurate and definite test of an increase of the intracranial pressure is the lumbar puncture, using the spinal mercurial manometer; in this manner, the ophthalmoscopic findings can be confirmed. Intracranial localization has been greatly facilitated by the most thorough neurological examinations, and yet in many cases the localizing signs are so obscured by the increased intracranial pressure that they can be easily overlooked and they may even be absent; the importance of examining these patients early is obvious. No patient should be allowed to develop a secondary optic atrophy and its resulting blindness while the effort is being made to localize the condition—an unimportant consideration in many patients; an early cranial decompression will save the eyesight and frequently the intracranial lesion can be localized later. Röntgen-rays are of much assistance in the diagnosis of many cranial and spinal lesions; by this means, long-continued intracranial pressure signs may also be determined and frequently the site of the lesion indicated; a negative picture, especially of the skull, however, means nothing, and frequently the interpretation of apparently positive plates is most difficult and at times confusing. Naturally, in cranial and spinal injuries, the value of the Röntgen-rays is very great indeed, especially regarding accurate diagnosis, and yet the treatment of such conditions, particularly of brain injuries, depends upon the presence or not of an increased intracranial pressure, whether the skull itself is fractured or not; whereas in spinal injuries, the chief concern is whether the spinal cord has been irreparably damaged or not—the spinal fracture being of little importance neurologically so far as the treatment is concerned, unless the vertebral dislocation is so extensive that the spinal cord must have lost its continuity.

An improved surgical technic, especially regarding the team-work of the operator and assistant, has been a large factor in lowering the mortality of neurological operations; not only is the rapid loss of blood avoided, the duration of the operation lessened and thereby the shock minimized, but the risk of infection is also proportionately diminished to a point practically nil. Naturally, intracranial operations should not be hurriedly done, but they can be quickly and at the same time smoothly and safely performed; there is surely no advantage to be obtained in prolonging the operation either on account of faulty technic or as a result of such a complicated technic that the final closure of the wound is delayed many minutes. It is rarely necessary for the team to consist of more than the operator, two assistants and a nurse; the anesthetist is a most important member and many disasters in cerebral surgery have been due to faulty anesthesia; Doctor Hunt's observations have resulted in his using a method of ether-oxygen that has proven most satisfactory during the last six years.

The third important factor in the progress of neurological surgery during the past decade has been due to a better understanding of the neurological condition at operation, both by the surgeon and by the neurologist at his side. Formerly, the surgeon knew little, if any, neurology, and the neurologist knew little, if any, surgery; the result was poor team-work and thus frequently the surgical judgment was not the best. A number of years ago, Dr. Allen Starr realized this so that in many patients he really performed the operation himself except the actual handling of the instruments; those operators who were not so fortunate to have a competent neurologist by their side groped along and frequently much damage resulted. To-day the surgeon should have at least a practical knowledge of neurological principles—both anatomically and physiologically; naturally, a training in neurological pathology is most essential. In this manner a number of mistakes in surgical judgment may be avoided; if the patient cannot be benefited, by no means make the condition worse by an operation.

One of the most important aids to an increased knowledge of neurological lesions, particularly of the brain and of the spinal cord, has been the observation of the living pathology at operation, and if death should occur, then the careful study of the tissue itself at autopsy. During the last five years, a permission for autopsy has been obtained before operation in each case of neurological surgery at the Polyclinic Hospital (both ward and private patients)—no operation being performed unless the permission is given in writing, so that if death should occur, then we shall ascertain its cause, and also the accuracy of the diagnosis and the treatment for the benefit of future patients. Naturally, when an operation is advised, it is in the belief that the patient will not die, but if the patient should die, then it is absolutely essential for the benefit of other patients that we ascertain the cause of death in order that possible similar mistakes, at least, may be avoided. I know of no means so enlightening to the doctor regarding the accuracy of diagnosis, particularly of intracranial conditions, than the post-mortem examination; besides the benefit of such knowledge to one's future patients, there is a marked tendency for these examinations to make the doctor humble as to his real knowledge and to keep him in that mental attitude.

If neurological surgery consisted chiefly of the removal of brain tumors, it would indeed be a most discouraging field of endeavor. Tumors of the brain are not only malignant in the vast majority of cases, but the diagnosis and accurate localization of cerebral tumors are often so delayed that the patients are frequently permanently impaired even after a surgically successful removal of the tumor; in addition to the mental and physical impairment, the danger of a secondary optic atrophy, with the varying degrees of impaired vision and even blindness, is a very common result of the prolonged increase of the general intracranial pressure. Naturally, the earlier a definite diagnosis of the tumor is made the better the prognosis. With the improved surgical technic and plan of attack in these patients, the operative mortality is low; when death does occur after operation, it is usually due to an attempt to do too much rather than perform a second stage operation at a later date. Besides these removable tumors of the brain, there are the so-called inaccessible tumors, situated at the base of the mid-brain; the pioneer work of Doctor Cushing in attacking the hypophyseal tumors by the sublabial, septal and transsphenoidal route is most brilliant; it is limited, however, to cases of primary pituitary tumor affecting the overlying optic chiasm and thus producing a primary optic atrophy; tumors of the mid-brain, which are much more common, cannot be approached by this method and it is rare that they can be successfully removed by any method now used; to offset their pressure effects by means of the operation of cranial decompression and thus delay the secondary optic atrophy is the most that can be hoped for in many patients.

There are few operations in surgery having the wide application and immediate beneficial results as the cranial decompression, and particularly the subtemporal method. It is an operation that has been very much neglected in the past and one that is capable of still greater usefulness in the future. It is a comparatively simple operation, requiring no special technic other than a thorough knowledge of the anatomy of the temporal region and the avoidance of operative complications; if, however, difficulties are encountered, then the use of the best methods for controlling them. Naturally, careful hemostasis is a most important factor in obtaining good results in all cranial operations; due respect and regard for the delicate nerve cells of the cerebral cortex by the avoidance of unnecessary and rough manipulation and digital examination of it; and, of the greatest importance, a most strict asepsis.

Cranial decompressions have been limited in the past chiefly to the relief of intracranial pressure in cases of unlocalized cerebral tumor, and in patients having a "fracture of the skull" and showing signs of medullary compression; the operation was performed not only to lessen the danger of a medullary edema, but to avoid a secondary optic atrophy so commonly observed in tumors of the brain. In these latter patients, the site of the decompression was most frequently over the parietal area or the upper temporal region, and thus, as the tumor enlarged, the increasing intracranial pressure forced the underlying cerebral tissue through the bony opening, producing a hernia cerebri of tremendous size—the bane of cranial surgery; a fungus cerebri was also a common result of such protrusion. Not only was this complication to be feared, but the operative danger to the underlying motor area with resulting paralysis of the opposite side of the body was always risked; besides, the intracranial pressure in "fractures of the skull," as well as in tumor formations, frequently produced a motor impairment by forcing the motor area upward through the bony ring of the decompression.

The reason for these complications is obvious: to remove an area of either parietal bone, not only may the underlying motor cortex be impaired at the time of the operation but also subsequently by its protrusion upward through the bony opening; this is made possible by the extremely weak protection afforded by the scalp overlying the parietal bone; besides the cutaneous tissues in this area, there is only the epicranial aponeurosis, so that even a moderate degree of intracranial pressure is sufficient to cause a hernial protrusion. If the decompression is performed in the lower parietal area, then the cranial origin of the temporal muscle to the parietal crest must be destroyed and thus the possible protection of the temporal muscle is lost.

In contrast to these methods of cranial decompression, the subtemporal route offers an almost ideal operation for intracranial conditions requiring either a relief of the increased pressure or an exploratory procedure; not only is the underlying cortex here a part of the temporo-sphenoidal lobe (which is a comparatively "silent" area of the brain), but the removal of the squamous bone is technically less difficult in that it is the thinnest part of the vault of the skull. Again, the decompression opening is amply protected by the overlying temporal muscle, so that it is a very rare occurrence to have a hernia cerebri following this method of cranial decompression; if the attachment of the temporal muscle to the parietal crest is carefully preserved, then it is practically impossible for a marked protrusion to occur; in my opinion, this method of decompression should be the one always to be employed. In subtentorial lesions affecting the cerebellum, naturally a suboccipital decompression is to be preferred; especially is this true of tumor and abscess formations in it. As the tentorium strongly separates the cerebellum, any increase of the subtentorial pressure is more effectively relieved by a suboccipital decompression than by a supratentorial operation; besides, not only may the lesion be removed at the same time, but the bony opening will be protected by the thick layer of occipital muscles and thus a large hernia be prevented.

The purpose of the subtemporal decompression has been very much enlarged during the past few years, and it seems that its usefulness is to be developed still more in the future. Although its chief function is the relief of intracranial pressure, yet it is a most valuable method of exploration. In these two divisions, practically all of the intracranial conditions for which the operation may be advisable, can be classified.

The operation of cranial decompression has been so frequently misinterpreted, and even confused with other cranial operations, that it seems advisable to state its indications and contra-indications, and to describe the type of decompression, with its most satisfactory technic.

As is indicated by the name of the operation, a cranial decompression presupposes the presence of an increased intracranial pressure; if there is not present an increased intracranial pressure, then the operation cannot be described as a decompression, but rather as a craniotomy (if the bone-flap is replaced), or a craniectomy (if the bone-flap is permanently removed). In the recent literature, the term decompression is commonly used to indicate merely a trephine opening, and in many instances the dura not even opened or allowed to remain open; surely a small trephine opening in itself with the dura opened may theoretically be to a certain extent, a decompression, but its decompressive effect is practically almost negligible; again, to replace a bone-flap even though the dura is allowed to remain open is not a decompression, because the decompressive effect of the operation is nullified. It would seem, therefore, that the three qualifications essential to a cranial decompression (in order that it be correctly considered as such) are:

1. The presence of an increased intracranial pressure.
2. The removal of a large area of the vault—usually 3 inches in diameter.
3. The dura to be opened and allowed to remain open; as the dura is inelastic in adults, no decompressive effect can be obtained unless the dura is opened, and if a permanent decompression is desired, then the dural opening must not be resutured but allowed to remain open.

Cranial decompressions were commonly performed in the past over the upper areas of the vault. Many disasters resulted from this procedure; in fact, the operation of cranial decompression for this reason was practically discredited and it remained an operation of the last resort until Cushing placed the operation upon a rational basis. In patients having a marked increase of the intracranial pressure as in brain tumor, not only was the operative damage to the underlying, highly developed cerebral cortex a frequent result of such decompressions situated over the upper portions of the vault and thus followed by paralyses, impairment of the special senses, and only too frequently the immediate death of the patient, but the insecure closure of the operative wound (merely covered by the scalp) permitted huge herniæ cerebri to occur and only too frequently their end-result, fungi cerebri and the death of the patient after months of a vegetative existence. It is no surprise therefore, that a few years ago the operation of cranial decompression was avoided as long as possible—even at the risk of the delay producing an impaired vision and even blindness itself, and it is this transmitted dread and fear of cranial operations that has retarded the development of brain surgery possibly more than any other factor. Cranial operations as now performed are no longer such extreme risks, while the operation of cranial decompression is in itself no greater risk than the usual abdominal operation. This advance is due chiefly to better team-work of the surgeon and the neurologist, a more practical conception of the purpose of the operation of cranial decompression, and then a most important factor, an improved surgical technic.

The operation of cranial decompression may be considered as a means solely of decompression, then of decompression plus exploration, and lastly, of decompression plus drainage. The following intracranial conditions as benefited by the operation of decompression may be classified briefly as follows:

- A. Decompression alone.
    - I. Brain tumors
        - 1. Irremovable tumors.
            - a. Large midbrain and basal tumors.
            - b. Large subcortical tumors, the removal of which would produce grave impairments, such as paralysis.
    - II. Selected cases of cerebral spastic paralysis due to an intracranial hemorrhage at birth.
- B. Decompression and exploration.
    - I. Brain tumors—non-localizable tumors, usually situated in the frontal and temporo-sphenoidal lobes.
    - II. Brain abscesses—non-localizable abscesses—usually situated in the temporo-sphenoidal lobes.
    - III. Selected cases of organic epilepsy—Jacksonian in type or associated with increased intracranial pressure.
- C. Decompression and drainage.
    - I. Brain injuries with or without a fracture of the skull.
    - II. Hydrocephalus—either of the internal or of the more common external type.
    - III. Brain abscess.
    - IV. Early localized meningitis especially due to otitic infections.

There are several other intracranial lesions for which the operation of cranial decompression has recently been advocated, notably the condition of apoplexy. It is possible in rare and selected cases of cortical apoplexy alone, and at times in ventricular hemorrhage (although the diagnosis and operative drainage should be almost immediate in order to have the patient survive) that the operation of cranial decompression and drainage might be considered in order to obtain the greatest ultimate improvement, but to advise this operation for the usual form of apoplexy—the internal capsular type, which occurs in 90 per cent. of these patients is a most unsurgical procedure; not only do these latter patients not have an increased intracranial pressure, unless the hemorrhage is of unusually large size, and therefore the operation described in the literature cannot be a decompression, but the operative damage resulting from the attempt (and it can only be a blind attempt) to insert a needle into the internal capsule and thus drain the hemorrhage (even if it would drain) would be far greater than the impairment caused by the lesion itself; besides, some of the reported patients have been operated upon late, when no drainage of the hemorrhage would be possible; and even in the early cases operated upon, it must be remembered that the hemorrhage into the internal capsule is rarely a free one, but is enmeshed among the capsular fibres, giving the appearance almost of liver tissue, and surely such hemorrhage cannot be drained. To advise, therefore, a cranial operation upon such patients merely because medical treatment has not caused a marked improvement is surely not rational surgery.

There may be selected cases of acute cerebral edema due to such toxic causes as occur in uremia and certain other toxic conditions where, after medical treatment has failed, a cranial decompression might be advisable to lessen a high intracranial pressure, and thus to a certain extent, spare the vision and avoid a medullary compression and its resulting edema—always fatal. No cranial operation, however, should be considered in these patients until repeated lumbar punctures have been proven inadequate. This work, however, is only in the experimental stage, and must be considered as a possible aid only in very selected patients.

The cranial decompressions of choice are the subtemporal decompressions for supratentorial lesions and the suboccipital decompressions for infratentorial lesions. Naturally, the subtemporal decompression as a decompression alone is of little or no benefit for infratentorial lesions and should never be used in such conditions unless as a means of ventricular drainage (in cases of ventricular blockage); infratentorial lesions such as cerebellar tumor and abscess are always more effectively treated by the suboccipital operation of decompression, exploration and drainage. In this book, however, the term cranial decompression means the subtemporal operation—the most satisfactory and effective method of decompression for supratentorial lesions; the vertical incision alone is used. (The advantages of this method of cranial decompression are detailed at the end of this chapter.)

Let us consider briefly the three main purposes of the subtemporal decompression:

A. Primarily as a Means of Lessening an Increased Intracranial Pressure.—I. *Brain tumors.* As the percentage of malignancy in brain tumors is high (almost 80 per cent.), they naturally form a very discouraging part of brain surgery; then again, if the tumor is not malignant in itself, yet it may be so situated in the mid-brain or base that to remove it would either cause the immediate death of the patient or such a marked mental, physical or sensory impairment that it would not be justifiable; that is, the condition of the patient might be worse than before the operation. It is not creditable to brain surgery to remove the tumor (even if benign) if the mental and physical condition of the patient is worse than before the operation—the patient remaining a derelict.

It is in these very patients that, in order to lessen the headache and to save the vision, an early subtemporal decompression on one side and, if necessary, on both sides of the skull is most strongly to be advocated. Not only will the general condition of the patient be improved and blindness avoided, but the tumor may not continue to enlarge and may remain stationary and even become smaller apparently, and thus the patient be spared indefinitely; this is particularly true in young adults—the diagnosis being a tuberculoma of the mid-brain; the edematous wet brain of the so-called pseudo-tumors might be also included. To allow these patients with a high degree of "choked disks" to develop a secondary optic atrophy and its resulting blindness merely because the tumor is considered an irremovable one or cannot be localized is an opinion that cannot be too strongly condemned. The operation of cranial decompression is no longer such a formidable procedure that it should be delayed and postponed until the life itself of the patient is endangered; to operate upon these patients blinded by long-continued intracranial pressure is most depressing and possibly hardly justifiable.

II. *Selected cases of cerebral spastic paralysis due to an intracranial hemorrhage at birth.* These patients were formerly classed in that large group due to Little's disease and thus confused with cases of lack of development of the cerebral cortex and its pyramidal tracts, and also with those cases resulting from a former meningo-encephalitis—an infectious destructive process and, naturally, conditions which cannot be benefited by any cranial operation. But patients having a spastic paralysis due to an intracranial hemorrhage at birth not only can be differentiated by the presence of an increased intracranial pressure as ascertained both by the ophthalmoscope and more accurately by a measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer, but those latter patients can be markedly improved by a lessening of this increased pressure by means of a simple subtemporal decompression. Naturally, only those patients having an increased intracranial pressure can be benefited and are therefore the only ones that are operated upon; out of a series now, January, 1918, of almost 1800 patients that I have personally examined, only 378 of them revealed the presence of an increased intracranial pressure—that is, about one out of every five patients examined—and these are the only ones for whom a cranial decompression can be of any benefit. The best results are obtained when the condition is diagnosed at the time of birth or shortly after it—and it very easily can be by the presence of blood under pressure in the cerebrospinal fluid at lumbar puncture; then, a modified cranial decompression with drainage of the free hemorrhage will in many cases obtain a normal child. (Even repeated lumbar punctures with drainage may suffice if the intracranial hemorrhage is not too large and the pressure too high; in these latter cases of high intracranial pressure the cranial operation of opening the parieto-squamous suture combined with a modified subtemporal decompression and drainage is advisable; the dura must always be opened.) The older the child the less is the improvement that can be ultimately obtained by the operation of merely lessening the increased intracranial pressure resulting from the earlier hemorrhage; frequently a mild secondary external hydrocephalus results in these patients from the blockage of the stomata of exit of the cerebrospinal fluid in the cortical veins and sinuses; this condition can be drained accordingly at the same time by a modified decompression operation (*vide* Hydrocephalus).

The pathology of intracranial hemorrhage in these patients is very instructive. It was formerly believed that the hemorrhage always caused a primary destruction of brain tissue and, therefore, no regeneration being possible, that these patients were all hopeless. As a permission for a post-mortem examination is obtained before operation upon each patient, it has been very surprising to ascertain, either at the operation or at autopsy, that the intracranial hemorrhage caused a primary destruction of brain tissue in only 26 patients out of 364 patients operated upon; that is, in only 7 per cent. The death-rate of these operations was only 38 out of the series of 374 operated patients—that is, a mortality of only 10+ per cent.—and a post-mortem examination was made in each case. The hemorrhage is almost always a supracortical one with later cystic formation, so that the mental and physical impairments in these patients are due to the increased pressure of the overlying lesion—both a general and a localized increase of the intracranial pressure. It is the lessening of this increased intracranial pressure in these selected patients that permits a marked improvement in them, both mentally and physically.[1]

diagnosed than the supratentorial temporo-sphenoidal ones, so that if cerebellar abscess can be excluded, then the operative approach of choice is through the "clean" subtemporal area and not through the "dirty" infected field of the mastoid incision; all drainage operations for brain abscess are really exploratory procedures, as the abscess may not be present or not found, and if the dural incision has been made through the infected mastoid area then the danger of causing a diffuse meningitis is very great indeed. If, however, the subtemporal exposure has been made and if the abscess is not found, then at least a decompression has been performed and the danger of a meningitis is slight; and if the abscess is found, then it can be freely and safely drained through the lower angle of the subtemporal incision. Not only does the decompression incision afford better drainage for the deeper subcortical abscesses by means of the double glass tubes, but the opportunity afforded of locating these deep abscesses is much greater and easier through the wider exposure of the decompression incision.[2]

III. *Selected cases of epileptiform convulsions—Jacksonian in type or associated with an increased intracranial pressure.* The surgical treatment of organic epilepsy is most discouraging, and no cranial operation should be ever advised in cases of long standing in the belief that the condition can be cured. In only very selected cases of short duration where the attacks are definitely of the Jacksonian localizing type and where there is a marked increase of the intracranial pressure (still persisting after an interval of at least three months has elapsed since the last attack), is the operation of cranial decompression and exploration or any cranial operation justifiable; even in the most carefully selected cases the results are not encouraging, and yet good result and apparently even a cure is occasionally obtained in these patients. Cranial surgery, however, cannot be said to offer much hope to these patients, except possibly to these very early selected patients as described above. To operate upon old chronic cases of whatever severity is hardly justifiable. Even the removal of the primary cause of the cortical irritation with its resulting convulsions—such as cortical tumor, old depressed fractures of the vault and the cyst formation due to a former supracortical hemorrhage, may result in only a temporary cessation of the convulsions in the older patients; but these patients should be diagnosed early, the irritating lesion removed and then the outlook is not so gloomy; besides removing the lesion itself, it is always wiser in these patients to relieve permanently the intracranial pressure due to the resulting cerebral edema of trauma by means of a subtemporal decompression.

C. **Decompression Plus Drainage.**—I. *Brain injuries with or without a fracture of the skull.* The mortality of brain injuries with or without a fracture of the skull has been notably decreased within the past decade; the former death-rate of 50 per cent. and even higher has been reduced in several hospitals to 30 per cent. and even lower. This marked improvement is due to a more general appreciation of the important factor of increased intracranial pressure in these patients and if present, then the most satisfactory method of relieving it by means of the subtemporal decompression.

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[2] *The Laryngoscope*, St. Louis, March, 1914.

As is well known, it is not the fracture in these patients, nor the hemorrhage in itself, that is so dangerous, but rather the presence of a high intracranial pressure—whether due to hemorrhage or edema, and its consequent medullary compression and the only too frequently resulting medullary edema—that renders these cases so serious—not only as to life but also as to their future normality.

Formerly, the treatment of these patients had been so discouraging that it became a commonly accepted belief that they would all "get along just as well without operation as with operation." This statement is perfectly true for over one-half of the patients—there being no increased intracranial pressure in practically this percentage, and naturally no operation (except in depressed fractures of the vault) would be indicated; but of the remaining patients upon whom a cranial operation was performed, the resulting high mortality—in many hospitals being 80 per cent. and even higher—was due chiefly to the operative method used—in almost all cases the extensive osteoplastic "flap" operation being performed and the dura very frequently not even being opened (and therefore no real decompression possible) or a small and totally inadequate trephine opening—the size of a one-half dollar piece and even smaller being considered as a sufficient operative procedure—these two operative extremes—the former tremendous operation of great risk to these seriously injured patients and the latter so-called operation of no possible, or if possible, then of very slight benefit to the patients—these two operations were usually performed either during the period of initial shock within a few hours after the injury, when it is now realized that no operation should ever be performed as it is merely an added shock to the patient and takes away the patient's chance of surviving the shock, or the operation was performed during the terminal period of medullary edema in the hope that the patient would be given a chance to recover; these patients when they have once reached this stage all die—operation or no operation. Naturally, under these conditions, the operative mortality was very high. More recent observations, however, in a series of almost 500 patients, have confirmed a growing belief in the following cardinal principles regarding the treatment of brain injuries[3] with or without a fracture of the skull:

1. All depressed fractures of the vault should be elevated or removed for fear of later complications, particularly epilepsy.

2. The presence of a fracture in patients having brain injuries is not an important factor in their treatment; the patients most seriously injured very commonly have no fracture at all, and conversely, the less serious of brain injuries are frequently associated with tremendous linear fractures of the vault—an important channel, however, of lessening an increased intracranial pressure by the drainage of free blood and excess cerebrospinal fluid or edema, and thus a cranial operation be avoided. Naturally, basal fractures into the middle ear and especially into the nasal and pharyngeal cavities may permit an infective meningitis, and yet this grave complication is comparatively rare.

3. The expectant palliative medical treatment of quiet, ice helmet,

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[3] *J. Am. M. Assn.*, May 13, 1916, lxvi, pp. 1536–1540.

catharsis and liquid diet is sufficient for over one-half of the patients having little or no increase of the intracranial pressure.

4. A marked increase of the intracranial pressure (as revealed in slightly over one-third of these patients by the ophthalmoscope and the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer) and *not* by the late and extreme pressure signs of medullary compression (such as a pulse-rate of 50 and lower, Cheyne-Stokes respiration and pulse, a high blood-pressure and profound unconsciousness) should be lessened by a cranial decompression and drainage—not necessarily at the site of the fracture if present, and by no means over the upper cortical areas, but rather by means of the subtemporal decompression—the safest and most effective means of decompression, drainage and closure possible in these patients. The dura must always be opened (except in the cases of simple extradural middle meningeal hemorrhage, which are rare) and allowed to remain open for a permanent decompression and drainage of the consequent traumatic cerebral edema so common in these patients. If the intracranial pressure is extremely high, then a bilateral subtemporal decompression and drainage may be necessary—apparently in about 5 per cent. of the patients.

5. There are two periods in the treatment of these patients when no operation is advisable, no matter how seriously the patient is injured nor how high the intracranial pressure may be; these periods are—first, the stage of initial shock immediately following the brain injury when the pulse-rate is 120 or higher; any operation during this period of traumatic shock is merely an added shock to the patient and takes away the patient's chance of surviving the shock; if, however, he does survive the shock, then he does so in spite of the operation; and the second period in which no operation should be performed is the stage of medullary edema—the terminal moribund period—operation or no operation; when once the pulse has reached its lowest level of medullary compression and then begins to ascend rapidly to 100, 120, 140 and higher, then an operation does not "give the patient a chance" (as formerly advocated), but merely hastens the exitus. If these two periods in the operative treatment of brain injuries are avoided and the latter of these—medullary edema—usually can be anticipated, then the mortality of brain injuries will be decreased to 30 per cent. and even lower, while the operation of decompression and drainage will make possible not only the recovery of patients as to life but also as to their future normality.

II. *Hydrocephalus, both of the internal and also of the more common external type.* A diffuse meningitis is the usual primary cause of this condition; if the ventricles are blocked by exudate or adhesions in the aqueduct of Sylvius or at the foramina of Majendie and Luschka, then the internal type of hydrocephalus results, but if this ventricular blockage does not occur, yet a hydrocephalus of the external type develops because the cerebrospinal fluid cannot be excreted as normally into the cortical veins and sinuses on account of their blockage by the former meningeal exudate.

In the operative treatment of hydrocephalus, either of the internal or of the external type, it is obvious that to drain merely the blocked cerebrospinal fluid of the ventricles into the subarachnoid or subdural spaces, by means of corpus callosal punctures or tubes, would, even if these openings remained patent, only change an internal hydrocephalus into an external one—with resulting little, if any improvement. The object, therefore, of all operative procedures in these patients is to drain continuously and permanently the blocked cerebrospinal fluid beyond the cerebrospinal canal—that is, into the blood stream (the ideal method and not yet practicable permanently) or into the extradural tissues, such as the subcutaneous tissues of the scalp, rich in lymphatics. The subtemporal decompression permits such drainage of the ventricles when blocked and of the subarachnoid and subdural spaces in the more common type of external hydrocephalus by means of several linen strands extending from the ventricles outward through the temporal lobe (a comparatively silent area), and from the subarachnoid and subdural spaces outward beyond the opened dura into the subcutaneous tissues of the scalp in a stellate manner.[4] At present, this method of drainage through the subtemporal decompression assures an excellent drainage to all but apparently the most severe types of complete blockage of internal hydrocephalus; naturally, the earlier the operation is performed following the development of the hydrocephalic condition, the greater is the chance of the child to approximate normality.

III. *Brain abscess.* As stated above, the usual type of brain abscess situated in the contiguous temporo-sphenoidal lobe following otitic disease is most safely and effectively drained through the lower angle of a "clean" subtemporal decompression by means of double glass tubes, so that the inner tube may be used to drain the abscess while the outer tube remains always *in situ*, and thus the abscess, when once found, is not lost. Apparently, a large decompression opening tends to lessen the danger of a complicating meningo-encephalitis—almost always fatal, whereas the opening through the "dirty" field of the mastoid not only increases the danger and lessens the opportunity of a careful exploration of the adjacent brain in search of the abscess, but it does not provide a satisfactory and efficient drainage for the deeper subcortical brain abscesses; again, to puncture the dura blindly with a knife through the mastoid opening or any dural exposure is most unsurgical.

IV. *Localized meningitis.* Similar to brain abscess formations, localized meningitis most frequently results from preexisting otitic and sinus disease; cranial fractures are also another common cause. Here again, in the early patients in whom lumbar punctures do not reveal the presence of an active organism in the cerebrospinal fluid and therefore indicating that the meningeal infection has not yet become a diffuse process, the operation of subtemporal decompression and drainage will offer a definite chance of recovery; although the prognosis is most unfavorable, it seems that in only the early and still localized conditions of purulent meningitis will the operation be of any benefit.

The technic of the operation of subtemporal decompression has been described briefly in the preceding chapter [illegible] and [illegible] the former method [illegible] is most satisfactory. The main advantages of the subtemporal decompression are the following:

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[4] [illegible]

1. It exposes, as widely as necessary, a comparatively "silent" area of the brain, the temporo-sphenoidal lobe, and therefore any operative damage to the exposed cortex will not appear clinically; also, in patients having a high intracranial pressure the danger of a hernial protrusion of a highly developed area of the brain with resulting paralysis, etc., cannot occur.
2. Being situated midway between the frontal and occipital lobes, it permits the careful exploration of all parts of the ipsolateral hemisphere; ventricular puncture, as well as permanent drainage, is also possible.
3. It exposes the area of the middle meningeal artery so frequently injured in the traumatic cases, and also affords excellent drainage to the middle cranial fossa at its lowest point—a very important factor in the treatment of brain injuries.
4. A firm closure of the decompression opening is obtained by means of the strong temporal muscle and its overlying fascia with their strong attachment to the parietal crest *intact*—a most important requisite in patients having a high intracranial pressure; hernial protrusions with their frightful fungi are most rare.
5. Technically, the operation is less difficult than other cranial operations in that the skull opening is made through the thinnest area of the vault—the squamous portion of the temporal bone.
6. The vertical incision is preferable to the former curved one in that it renders more possible a careful hemostasis of the scalp by means of the method of bi-manual pressure-traction and the clamping of the main branch of the temporal artery at the very beginning of the operation, whereas the curved incision passes through the various branches of the vessel in the scalp and they must be clamped individually; again, the vertical incision not only permits drainage at the lowest point of the skull, but it makes possible a large subtemporal bony opening without risk of loosening the attachment of the temporal muscle and fascia to the parietal crest, and thus a firm closure with no danger of cerebral hernia is assured.
7. The great frequency of temporo-sphenoidal lesions such as tumors, abscesses, and brain injuries make this routine exposure of the subtemporal decompression a most important aid in the treatment of underlying intracranial lesions.

Conclusions

.The operation of cranial decompression is one that should be used much more frequently than it is at present; especially is this true in the conditions of brain tumor, brain abscess, brain injuries and in selected cases of spastic paralysis due to an intracranial hemorrhage at birth.

The subtemporal method of cranial decompression is the ideal route; besides being less difficult technically, it exposes an area of the brain most frequently involved. This permanent decompression opening does not weaken the skull in that the thick overlying temporal muscle protects it most adequately, so that hernial cerebri are not to be feared.

The operative mortality is low. Patients with intracranial conditions should not be permitted to become blind or to reach the dangerous stage of medullary compression without a subtemporal decompression being performed early. (See Plates I–VIII.)

MOVING PICTURES OF THE OPERATION OF RIGHT SUBTEMPORAL DECOMPRESSION AND DRAINAGE

The patient was an alcoholic cab-driver of forty-six years of age, who was brought to the hospital in the ambulance following a fall from his driver's seat; unconscious; profuse discharge of blood and cerebrospinal fluid from the right ear. Repeated examinations disclosed the signs of a high intracranial pressure as registered by the ophthalmoscope and the spinal mercurial manometer and also by the gradual descent of the pulse- and respiration-rates, so that a right subtemporal decompression and drainage was performed ten hours after the injury. An irregular linear fracture of the right squamous bone had torn the right middle meningeal artery, causing a small extradural hemorrhage; the dura was tense and bluish, and upon incising it, a large amount of supracortical hemorrhage escaped, permitting the bulging cerebral cortex to recede and to pulsate normally. Usual closure with drainage. Excellent recovery.

THE INCISION OF THE SCALP; METHOD OF ASEPSIS AND HEMOSTASIS

A. Outlining the superficial skin incision from the zygoma upwards to the parietal crest—and not beyond. The head has been prepared in the usual manner for the operation; ether-oxygen apparatus in position.

B. Two layers of sterile gauze placed over the operative area and the sterile towels fastened to the scalp by the towel clips.

C. Cutting the sterile gauze layer with scissors and exposing the scalp incision only—and not the surrounding scalp.

D. Field of operation prepared with the scalp incision exposed. The danger of infection is now practically *nil*.

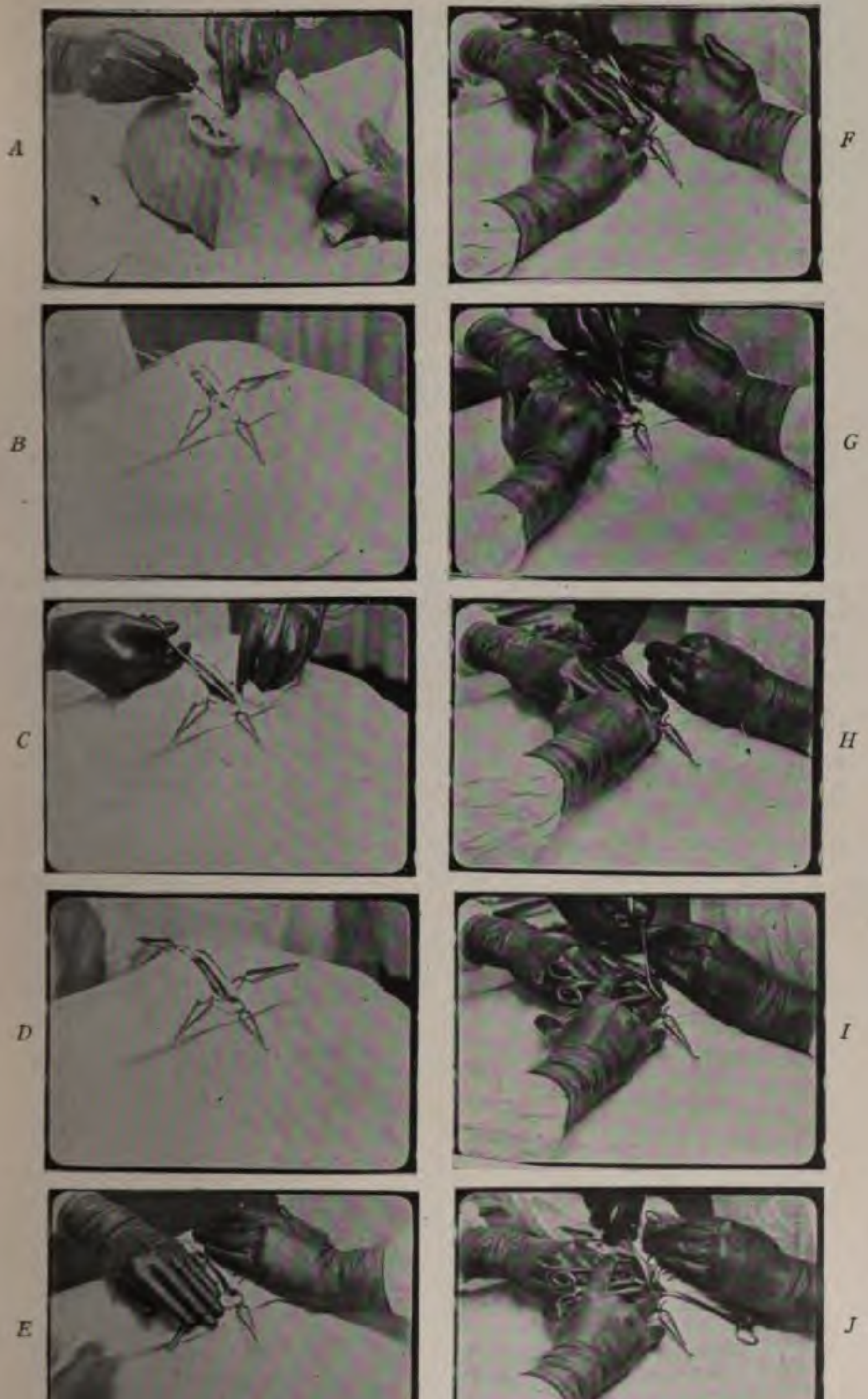
E. Applying the bimanual-pressure-traction method of hemostasis: the left hand of both the operator and his assistant used to compress the scalp vessels upon each side of the incision, and the assistant's right hand used to compress the temporal artery at the lower angle of the incision as shown in the following picture.

F. Using the scalpel to make the scalp incision down to the temporal fascia—the method of hemostasis by bimanual-pressure-traction being shown.

G. Scalp incision enlarged; also a small opening in the underlying temporal fascia permitting the temporal muscle to protrude. Note the excellent hemostasis obtained.

H, *I*, and *J*. Applying the small curved hemostats to the subcutaneous fascia for the compression of the scalp vessels; the turning-back of the hemostats affords excellent hemostasis upon the removal of the manual pressure.

PLATE I



OPENING THE BONE THROUGH THE TEMPORAL FASCIA AND MUSCLE

A. Continuing to apply the small curved hemostats to the subcutaneous fascia for the compression of the scalp vessels until no bleeding occurs upon the removal of the manual pressure.

B. Incising the temporal fascia from the zygoma vertically upwards to its attachment to the parietal crest—but not beyond.

C. Separating the fibres longitudinally of the underlying temporal muscle and exposing the underlying bone.

D. Inserting the two lateral retractors of the temporal muscle and fascia in order to expose the largest area possible of the underlying bone. (Care must be used not to sever the attachment of the temporal fascia and muscle to the parietal crest and thus weaken the closure.)

E, and *F*. The sharp periosteal elevator being used the remove the so-called periosteum overlying the bone. A small portion of the line of fracture can be seen.

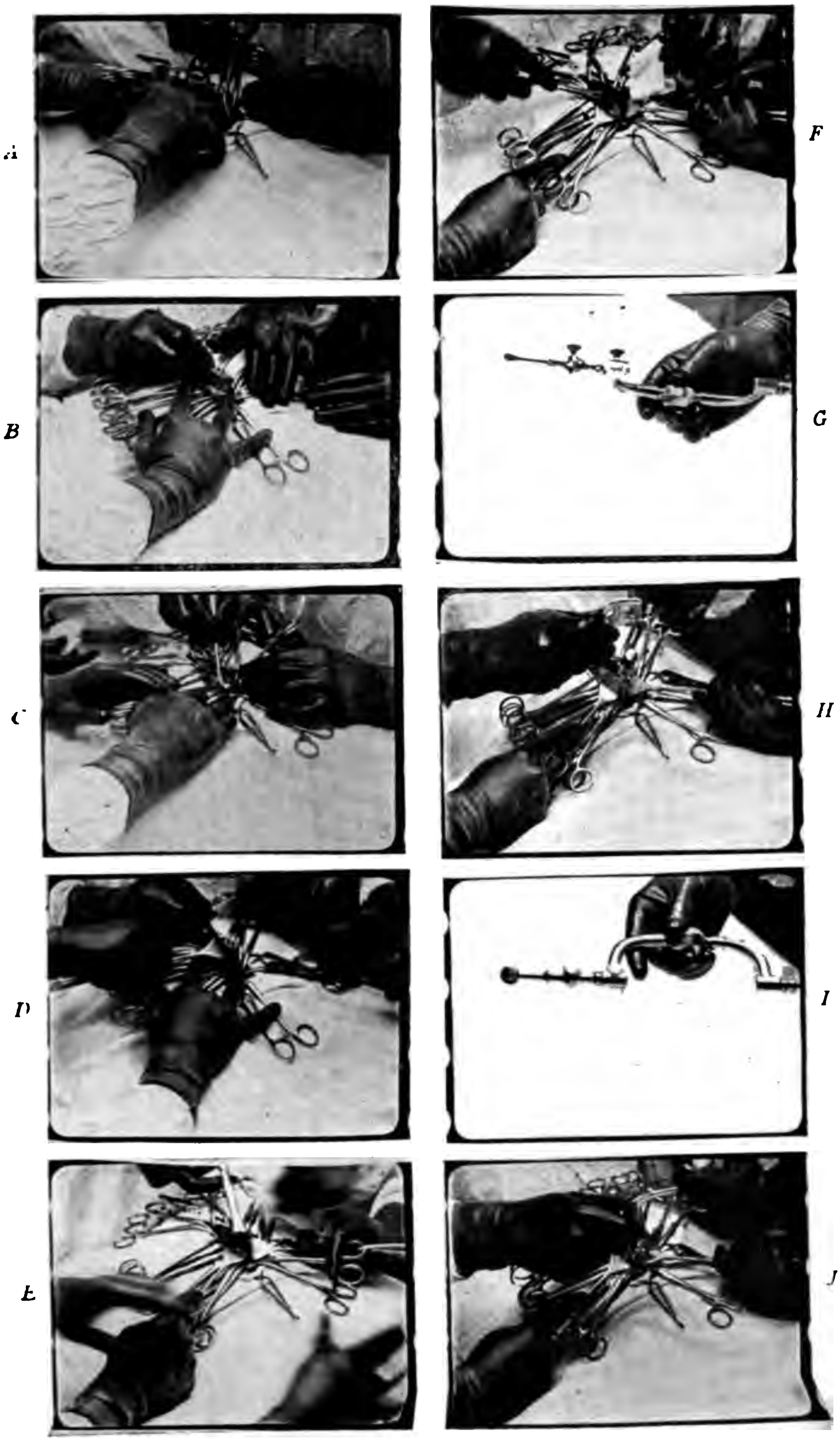
G. The Doyen perforator with its pointed tip to be used to make a conical opening of the bone down to the dura.

H. Using the Doyen perforator to make the bony opening at the lower angle of the incision—the thinnest portion of the squamous bone.

I. The Doyen burr to be used to enlarge the bony opening for the insertion of the small rongeurs.

J. Using the Doyen burr which enlarges the outer portion of the perforator opening only and does not penetrate deeper.

PLATE II



RONGEURING THE BONY OPENING

A. The small pin-point opening of the bone exposing the underlying dura; the middle meningeal artery can thus be easily avoided. The irregular line of fracture is clearly seen.

B, and *C*. The small pointed rongeurs being used first to enlarge the bony opening downwards and backwards (away from the middle meningeal artery).

D. The larger rongeurs with the lower blade thinned and bevelled so that it can be inserted safely between the bone and the underlying dura.

E. Using the larger rongeurs to enlarge the bony opening as widely as possible beneath the temporal muscle.

F. The dural separator to be used in severing adhesions between the dura and the overlying bone so that the dura will not be torn by the rongeurs.

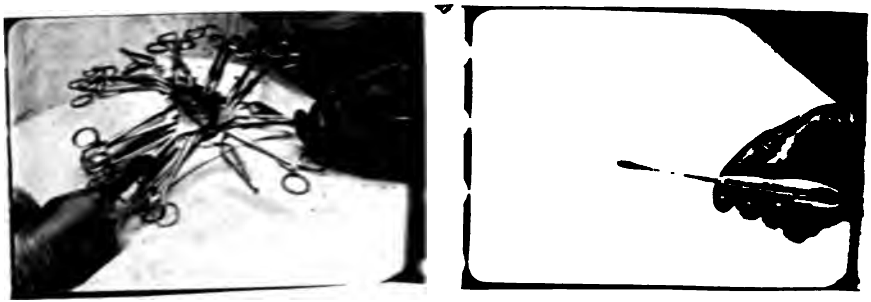
G. Using the dural separator to insert a small gauze tape between the dura and the overlying bone and thus compressing a bleeding dural vessel.

H. Another view of the larger rongeurs showing the thinned and bevelled lower blade.

I. Enlarging the bony opening downwards and backwards by the larger rongeurs.

J. A view of the bony opening at this stage of the operation; a small extradural blood-clot can be seen protruding downward from under the upper edge of the bone.

PLATE III



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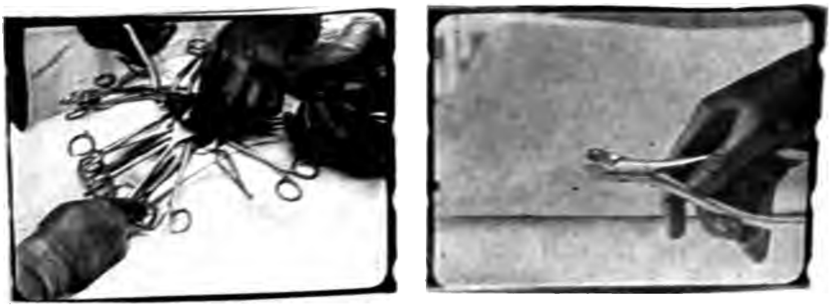


D

E

ENLARGING THE BONY OPENING; THE USE OF BONE-WAX

A. Continuing to remove the bone as much as is possible beneath the temporal muscle.

B. A pellet of sterile bone-wax to be used to stop any bleeding from the diploetic vessels by its being rubbed into the bony edge; this wax is an excellent hemostatic.

C, and *D*. Rubbing the bone-wax into the bony edges and thus causing all bleeding to cease.

E. Continuing to enlarge the bony opening by using the large rongeurs.

F. A view of the bony opening at this stage of the operation; it is necessary now to remove the bone of the upper portion of the operative field. The dura is seen to be tense and bulging and therefore under pressure.

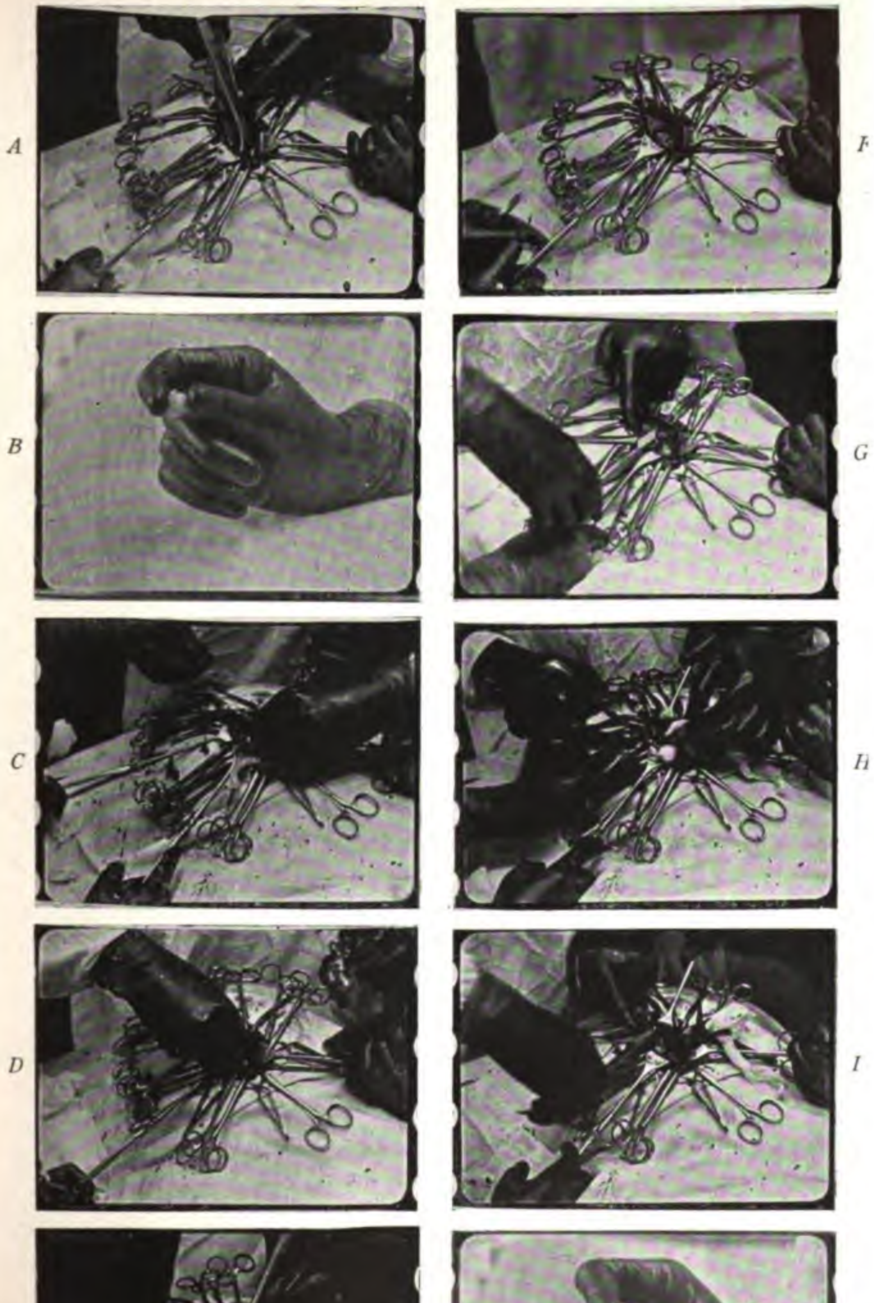
G. Using the large rongeurs to remove the bone along the line of fracture and overlying the middle meningeal artery.

H. Upon removing a bony fragment compressing the meningeal artery, t ing from this torn vessel was profuse, requiring the insertion of two strips gauze between the bone and the artery proximal to the bleeding point.

I. The torn middle mengineal artery having been successfully compressed by the small gauze tape, the rongeurs are again used to enlarge the bony opening.

J. Another view of a pellet of bone-wax illustrating its soft gummy character.

PLATE IV



EXPOSURE OF THE DURA

A. Rubbing the bone-wax into the diploë of the bony edge to control the oozing of blood.

B. Another view of the dural separator.

C. Using the dural separator to insert a small gauze tape between the dura and the bone at the lower angle of the operative field for the control of meningeal bleeding.

D, and *E*. Using the dural separator to sever adhesions between the dura and the overlying bone so that the danger of tearing the dura with the rongeurs is practically *nil*.

F, and *G*. The large rongeurs enlarging the bony opening as much as possible beneath the temporal muscle—to a diameter in adults of almost three inches.

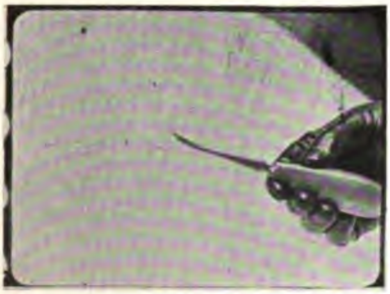
H. Rongeuring the bone downward as low as possible to the base of the skull, so that the drainage of subdural blood and excess cerebrospinal fluid in the middle fossa is facilitated.

I. The tense and slightly bluish dura bulging into the large bony opening; slight bleeding from a meningeal vessel being controlled by a small cotton pledget wet in warm normal saline solution.

J. The small dural hook which is used to elevate the dura when being incised and thus preventing any damage to the underlying cerebral cortex.

PLATE V

A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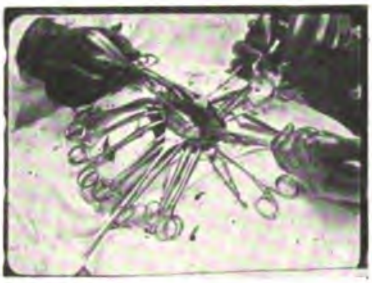

I

J

OPENING THE DURA

A. Making a small dural incision by elevating its outer layer with the dural hook so that the inner layer of the dura can then be safely and easily opened.

B. The small dural opening being enlarged by incising the dura upon a grooved director bent almost to a right angle: a small amount of subdural blood is seen escaping.

C. The smooth, flexible spoon-spatula for insertion beneath the dura so that the underlying cerebral cortex is not damaged by the dural scissors.

D, and *E*. Enlarging the dural opening as widely as possible with the dural scissors—the spoon-spatula being inserted beneath the dura.

F. A small V-shaped silver clip in its grooved holder for the clamping of a meningeal vessel before it is severed; in this manner, the dural opening is safely enlarged with no loss of blood.

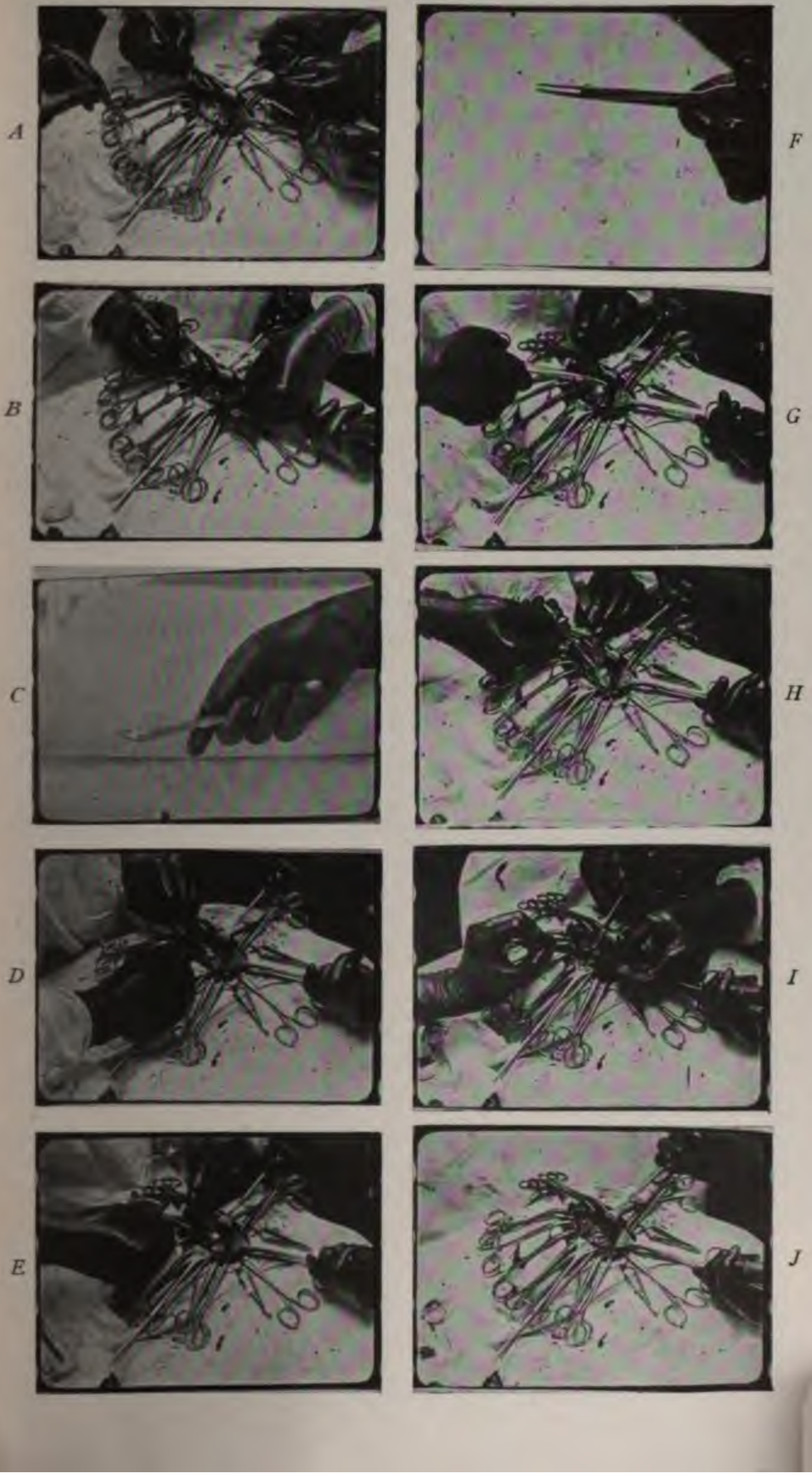
G. Applying the silver clip to the proximal portion of a meningeal vessel to be severed.

H. After a second silver clip has ligated the meningeal vessel at a point distal to the first clip, then the dural scissors sever the vessel between the two clips—and no bleeding occurs; the spoon-spatula protects the underlying cerebral cortex from all possible damage.

I. Enlarging the dural opening upward by means of the dural scissors the spoon-spatula.

J. The tense bulging cerebral cortex tending to protrude through the dural opening; a large quantity of supracortical hemorrhage and excess cerebrospinal fluid is escaping so that slight cerebral pulsation is now visible.

PLATE VI



CLOSURE OF OPERATIVE INCISION

A. Applying a silver clip to a troublesome bleeding vessel in the lower angle of the dural opening. Closure of the incision should not be started until all bleeding vessels have been controlled and a small drain of rubber tissue inserted intradurally at the lower angle of the incision into the middle fossa beneath the temporo-sphenoidal lobe.

B, and *C*. The split temporal muscle being sutured together in one layer over the cerebral cortex by continuous catgut (No. 2); another drain of rubber tissue is inserted intradurally at the upper angle of the incision. The dura is naturally not sutured.

D, and *E*. The temporal fascia being sutured by interrupted catgut (No. 2) alternating with interrupted sutures of fine black silk (waxed).

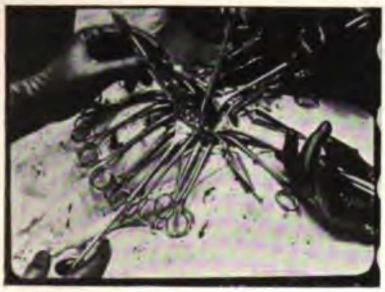
F. The subcutaneous fascia being sutured by interrupted catgut (No. 1); as these sutures are tied, the adjacent hemostats can be removed with little or no bleeding from the compressed vessels of the scalp. (The operative towels and sheets are observed to be only slightly soiled by blood.)

G, and *H*. Inserting and tying, respectively, the interrupted fine black silk sutures (waxed) of the scalp itself.

I. Approximating closely the upper layers of the epithelium of the scalp with interrupted fine black sutures of waxed silk.

J. The scalp incision closed and the towels and head drapery removed; a drain of rubber tissue is seen extending from both the lower and the upper angle of the incision.

PLATE VII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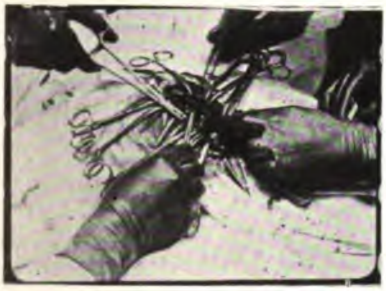
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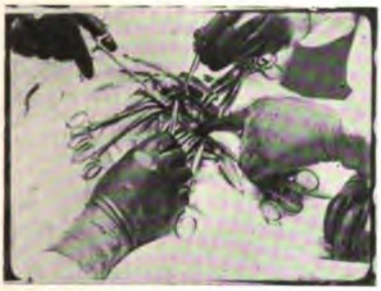
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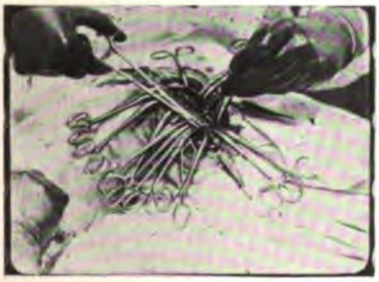
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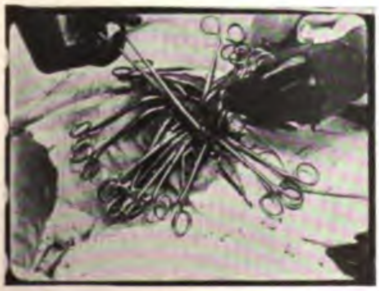
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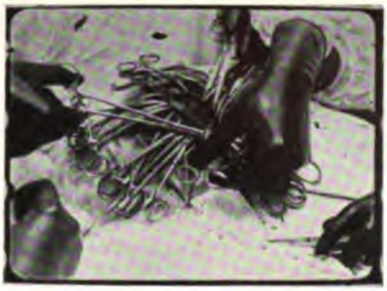
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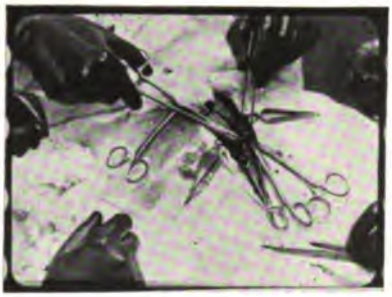
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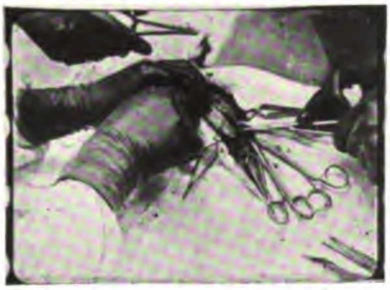
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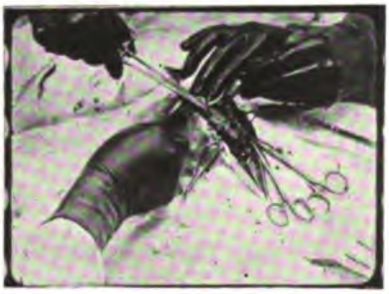
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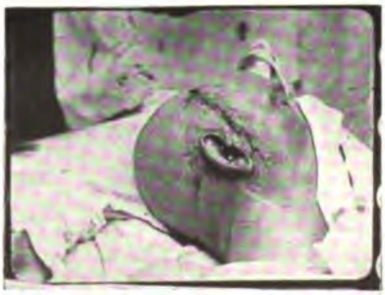
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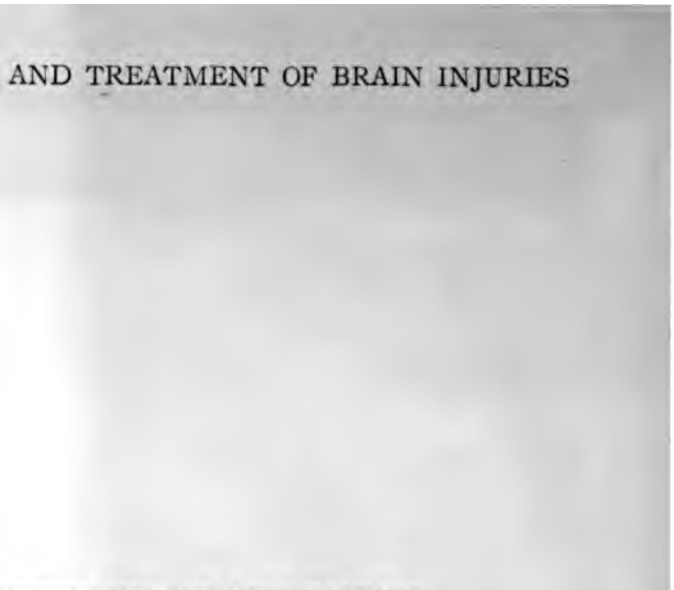


THE DRESSING AND HEAD BANDAGE

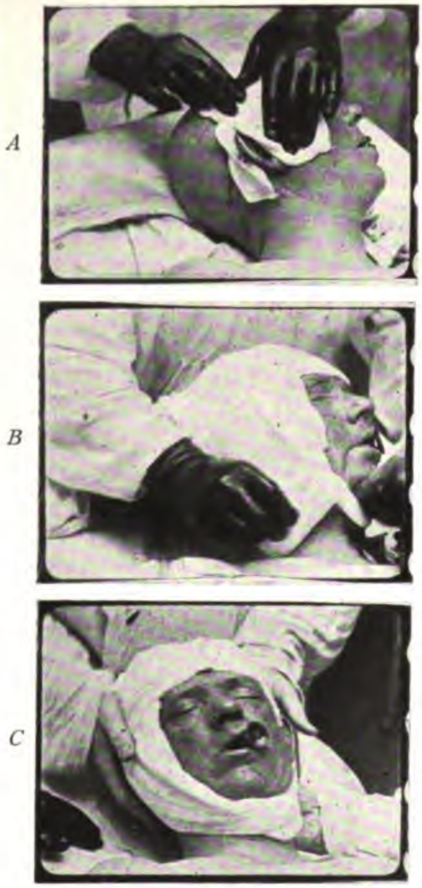
A. After the scalp incision has been gently sponged with a sterile gauze moistened in alcohol, a dry sterile gauze pad is placed over the operative area. Another small sterile gauze pad smeared with sterile vaseline is inserted behind the lobe of the ear to prevent its painful compression by the head bandage.

B. A sterile roller gauze bandage being applied about the head and under the chin; the opposite ear is not covered. Two fingers inserted beneath the chin preve the head bandage from being too snugly applied as to interfere with respiration.

C. Several strips of adhesive plaster used to "anchor" the gauze bandage so t it is held firmly in position. The uncovered ear of the opposite side of the head visible; also the laryngeal tube which facilitated the administration of ether in t patient and lessened the cyanosis

ATION OF CRANIAL DECOMPRE

PLATE VIII



PART II

ACUTE AND CHRONIC BRAIN INJURIES IN ADULTS. ILLUSTRATIVE CASES

CHAPTER X

ACUTE BRAIN INJURIES

THE case-histories reported in this chapter are from a series of patients having acute brain injuries; all of the patients have been classified according to the diagnosis, pathology, treatment, and their present condition; if death occurred, then the autopsy findings. Each patient has been traced in detail from the time of the cranial injury until the present time and the present condition of recovery, both of life and of the patient's former good health, has been recorded; the "bad" results are detailed as well as the "good" ones, and a critical attitude of the treatment of each patient is maintained. The autopsy findings have been most instructive; the mistakes of diagnosis and treatment thus ascertained tend to prevent their repetition, —at least, their frequent repetition.

Under this heading are included not only those patients in whom a definite change of cerebral tissue has occurred as the result of a recent cranial injury and due to cerebral lacerations, hemorrhage or edema, but also those acute cases of cranial injury which, by their clinical history and confirmed by repeated neurological examinations, can be classified as simple concussion, severe concussion and concussion complicated by various factors; post-traumatic neuroses; cranial injuries producing a doubtful fracture of the skull; various types of fractures of the vault; then finally that large group of patients in whom a definite intracranial lesion has occurred following the cranial injury with and without a fracture of the skull; autopsies were performed upon those patients who were unable to recover from their injuries. Naturally, the patients in whom there was no intracranial lesion of hemorrhage and edema sufficient to increase the intracranial pressure to a degree dangerous to life and to future normality, these patients were not operated upon—almost two-thirds of the patients, whereas if the intracranial pressure increased beyond a degree commensurate with life and future good health, these patients were operated upon—about one-third of the patients (31 per cent.); the patients having depressed fractures of the vault were all operated upon and the depressed area of bone either elevated or, more frequently, removed.

At the end of each case, certain points of interest regarding the diagnosis and treatment are discussed; where mistakes have occurred, either through carelessness or ignorance, an effort has been made to recognize them, while mistakes of judgment are pointed out in the hope that a similar error will in the future be avoided; the condition of the patients years after the injury, the operative findings, and especially the autopsy findings with and without a preceding operation should and do impress one with the fallibility of our present knowledge of intracranial lesions and their treatment, and particularly is this true of brain injuries; it should and does stimulate us to exert every effort to ascertain the actual intracranial condition by every method now known in order that the appropriate treatment be instituted early.

Cerebral Concussion

If the term "concussion" is limited to the result of those cranial injuries in which merely a sudden jarring of the intracranial contents—the old "*commotio cerebri*"—produced a temporary functional impairment of the brain and naturally without any ascertainable change of tissue, then the use of the term "cerebral concussion" is probably correct clinically. The condition itself, however, of simple cerebral concussion, as above restricted in its application, is of comparatively infrequent occurrence in hospital practice, although it is a term most freely used and carelessly applied; the more modern methods of examination, and especially the use of the lumbar puncture, have made it possible to ascertain more accurately the true intracranial condition in these patients, and it is surprising how frequently latent brain injuries masquerade under this term. A definite increase of the intracranial pressure as recorded by the ophthalmoscope and the spinal mercurial manometer and indicating an edematous condition of the brain, and surely the presence of blood in the cerebrospinal fluid, point to a condition of greater severity than a simple concussion.

Possibly the "knockout" in a boxing contest is the best example of a simple and true condition of cerebral concussion; that is, the defeated boxer receives a blow from his opponent's fist upon the lower jaw—the force of the blow being transmitted upward, either by the ramus on the same side as the point of contact and occurring in an "upper-cut" or by the ramus of the opposite side of the jaw and following a "swing"; an "upper-cut" delivered directly upon the point of the jaw and in the mid-line may have its force transmitted to the skull and the intracranial contents by both rami of the jaw; either or both mandibulo-temporal articulations are usually painful and tender. As the result of this sudden "jolt," consciousness is temporarily lost—possibly not longer than twenty seconds, and rarely persisting more than several minutes; the pupils are slightly enlarged and react to light sluggishly and the blood-pressure may be 110 and even lower. Upon the return of consciousness, the man quickly recovers from the stupor, complains of a dull headache which may continue for twelve to thirty-six hours and then he is apparently "as well as ever." It has been very interesting to observe, however, in two fighters who have been "knocked out" several times that both of them frequently complained later of headache during a period of months and each of them was so susceptible to a blow upon the chin or even the head itself that a "knockout" easily resulted—in fighting parlance, a "tin and glass jaw." Upon more careful examination, each of these men had a chronic edematous condition of the brain—the reflexes exaggerated but no Babinski, while the nasal margins of both optic disks were indistinctly blurred in the presence of enlarged retinal veins, and the spinal mercurial manometer registered the slightly increased pressure of 11 mm.; no neurotic or psychic factor could be ascertained in either of these men: naturally, any cardio-vascular or nephritic complication had also been excluded. It would seem that this post-traumatic cerebral edema lessened the resistance of these men to any blow upon the head which aggravated the existing "wet" condition of the brain and it was obligatory for them to [illegible] their "ring" activities.

Discharged April 23, 1914—2 days after injury.

Family history negative.

Personal history negative.

Present Illness.—Patient was "beaten up" in a fight in the new subway; taken to the police station, where he remained all night in a semi-conscious condition; brought to the hospital this morning at 7 o'clock.

Examination upon admission (7 hours after injury).—Temperature, 98.6°; pulse, 92; respiration, 24; blood-pressure, 128. Mild degree of shock. Semi-conscious. Bleeding from right ear; otoscopic examination revealed abrasion of the anterior wall of the external auditory canal; tympanic membrane normal. Tenderness just anterior to right ear and posterior portion of zygoma. Contusion of upper lip. Contusion of left thigh. Pupils equal and of normal reaction. Reflexes, present and equal; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi negative. Lumbar puncture—cerebrospinal fluid clear and under normal pressure (approximately 8 mm.).

Treatment.—Expectant palliative treatment of quiet, external warmth to body, soapsuds enema and ice-helmet.

Examination (5 hours after admission).—Temperature, 99°; pulse, 86; respiration, 22; blood-pressure, 144. Patient sleeping quietly; easily aroused. Mentally confused; complains of occipital headache; otherwise negative. No bleeding from ear. Reflexes negative. Fundi negative. Liquid diet.

Examination at discharge (one day after admission).—Temperature, 98.6°; pulse, 84; respiration, 20; blood-pressure, 140. Patient refuses to remain in the hospital longer. Slight general headache; emotionally irritable. Mentality clear. Reflexes negative. Fundi negative.

Examination (October 26, 1915—18 months after injury).—No complaints. Physical examination negative.

Examination (July 10, 1918—51 months after injury).—No complaints. Physical condition negative.

Remarks.—It is surprising how quickly patients recover from the condition of simple cerebral concussion uncomplicated by shock of any severity; it is not unusual for these patients immediately following an injury to appear to be most seriously injured—a question of life itself, and yet within 18 to 24 hours later the condition has so improved that the patient may be considered to be out of danger. As most head injuries, however, are associated with a greater or less degree of shock, then the recovery is not so rapid; if the shock is extreme, a marked improvement may be delayed for several days; naturally the treatment in these latter patients should be directed toward the condition of shock, and as it improves, then the signs of mild or severe cerebral concussion will appear; it is not at all unusual after the shock has subsided to ascertain in the patients having a severe condition of concussion, mild signs of an increased intracranial pressure—but of only short duration and rarely lasting longer than 12 hours.

It would seem that in many of these patients having cerebral concussion that a mild edematous condition of the brain occurred for several hours following the cranial injury and that this mildly "wet" condition does not last longer owing to the excellent vascular reaction that occurs, particularly in youthful adults; the older the patient is above 40 years of age, the less rapid and the less able is the patient to react to this condition and naturally the convalescence is lengthened. It is very difficult to differentiate at times the conditions of simple, severe cerebral concussion from traumatic cerebral conditions of greater severity and frequently the condition must be observed over a period of days and even weeks before the diagnosis of simple cerebral concussion can be made with certainty; if the condition persists longer than four days to one week following the injury, then the diagnosis must be very guarded in that the intracranial condition may be one of more than merely simple cerebral concussion; if there is any doubt at all, the most accurate examinations and tests should be repeatedly used in order to avoid if possible future complications.

The mild degree of shock present in this patient upon admission must have been of greater severity during the preceding seven hours while lying in the station-house; and yet within five hours after admission to the hospital and merely with the routine expectant palliative treatment of quiet, external warmth and ice-helmet, the condition of the patient so improves that his blood-pressure has risen from 128 to 144 and the pulse and respiration have both begun to descend slightly—favorable prognostic signs.

The value of otoscopic examinations in the presence of merely blood escaping from the external auditory canal is illustrated in this patient; with the improved electrical otoscopes and a sterilization of the otoscopic speculum, the danger of introducing infection into the inner portion of the external auditory canal is very slight indeed, and it is not necessary to insert the speculum farther than just within the external auditory meatus. If it had been ascertained at this examination that the tympanic membrane itself had been ruptured, then the patient would have been "watched" even more carefully for fear that a more serious intracranial lesion would develop—now that it was known that the base of the skull had been fractured; and there would also be the increased danger, although slight, of infection entering through the middle ear. However, it having been ascertained that the discharge of blood from the right auditory canal was due merely to an abrasion of the canal wall itself, and in the absence of marked signs of increased intracranial pressure, the prognosis could be considered good and the expectant palliative treatment advised with assurance.

II. Severe Cerebral Concussion

Case 2.—Severe cerebral concussion; fracture of surgical neck of left humerus. Excellent recovery.

No. 77.—Rose. sixty-eight years. White. Married. Housework. Russian Jewess.

Admitted November 17, 1914, Polyclinic Hospital. Referred by Doctor John A. Bodine.

Discharged December 14, 1914—27 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While crossing the street, patient was struck by an automobile and thrown upon the cement; brought to hospital in the automobile.

Examination upon admission (18 minutes after injury).—Temperature, 98.2°; pulse, 90; respiration, 30; blood-pressure, 122. Patient was conscious but dazed. Severe degree of shock. Slight laceration of left forehead and moderate contusion; marked contusion of left shoulder; definite crepitus, false motion and sharp pain in left shoulder upon manipulation of left arm. Moderate contusion of left hip. No bleeding from nose, mouth or ears; no mastoid ecchymosis. No facial paralysis. Pupils enlarged equally and react to light sluggishly. Reflexes depressed but equal; no Babinski. Fundi negative.

Treatment.—Expectant palliative treatment; external warmth. Arm not treated until shock of accident had disappeared.

Examination (8 hours after admission).—Temperature, 98.8°; pulse, 78; respiration, 22; blood-pressure, 148. Mentally confused; complains of severe frontal headache and pain in left shoulder. Pupils negative. Reflexes negative. Fundi negative. X-ray (Dr. A. J. Quimby): "Skull negative. Left shoulder revealed a fracture dislocation of surgical neck of left humerus."

Treatment.—Appropriate position of abduction and plaster cast for shoulder and arm applied. Back rest and frequent turning from side to side for fear of pneumonia. Liquid diet.

Examination (4 days after admission).—Slight headache and aching pain in left shoulder; otherwise negative. Reflexes negative. Fundi negative.

Examination at discharge (27 days after admission).—Temperature, 98.4°; pulse, 78; respiration, 22; blood-pressure, 146. No complaints except soreness in left shoulder. Pupils negative. Reflexes negative. Fundi negative.

Examination (May 10, 1915—5 months after injury).—No complaints except those incident to old age. Reflexes and fundi negative.

Last Examination (April 3, 1918—55 months after injury).—No complaints referable to head injury. Reflexes and fundi negative. Patient cannot be found since this examination.

Remarks.—Youthful adults withstand head injuries, and particularly the mild results of them, such as concussion, much better than do patients over forty years of age. There is less danger of the mild cerebral edema being prolonged in that these youthful patients react much more quickly than older ones. If this cranial injury had occurred to many patients over middle age and especially if they were addicted to alcohol or if their resistance had been lowered by any cardio-vascular or cardio-nephritic lesion, then the prognosis would undoubtedly have been very grave; it is rare for these latter patients to recover entirely from a severe cranial injury—even though the diagnosis is only "concussion," so that they are rarely just as well five years after the injury as before the injury.

Cranial injuries occurring to patients over sixty years of age are always serious even if the resulting intracranial condition is diagnosed as simply a "concussion" of varying degree; the danger is not so much one of intracranial complications as it is of, in the first place, hypostatic pneumonia, cardio-vascular and then cardio-renal disturbances; if the patient has been at all alcoholic, then that factor is of great significance. Obese patients do not withstand the effects of cranial injuries nearly so well as people of normal weight, and even, it would seem, of under weight.

III. CEREBRAL CONCUSSION AND ITS MOST FREQUENT COMPLICATIONS

A. *Extensive scalp lacerations.*

CASE 3.—Cerebral concussion; extensive laceration of scalp. Pott's fracture of left ankle. Recovery.

No. 732.—Kate. Fifty-six years. White. Single. Designer. U. S.

Admitted November 14, 1916, Polyclinic Hospital.

Discharged December 22, 1916—38 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While crossing the street, patient was hit by an automobile and knocked down, receiving an extensive laceration of scalp, bruises all over the body and fracture of left ankle. Recovered consciousness in the ambulance.

Examination upon admission (43 minutes after injury).—Temperature, 98°; pulse, 110; respiration, 32; blood-pressure, 118. Patient in considerable shock; semiconscious. Deep laceration of scalp of 4 inches in length over frontal region. Swelling of left ankle painful; no bleeding from ears, nose or mouth; no mastoid ecchymoses. Pupils equal, but are slightly dilated and react sluggishly. Reflexes difficult to obtain; abdominal reflexes absent. Fundi negative.

Treatment.—On account of the degree of shock, a warm wet bichloride dressing (1–5000) applied to laceration of the scalp, and the left ankle "just let alone." Expectant palliative treatment of external warmth and quiet.

Examination (6 hours after admission).—Temperature, 98.8°; pulse, 88; respiration, 24; blood-pressure, 132. Conscious but rather confused mentally. No mastoid ecchymosis. Pupils equal and react normally. Reflexes present and equal. Fundi—slight dilatation of retinal veins but no edema of the margins of the optic disks. Lumbar puncture—cerebrospinal fluid clear and under normal pressure (approximately 9 mm.). Scalp laceration examined; probe reveals no fracture of the underlying bone, wound cleansed with iodine and loosely sutured; 2 rubber tissue drains inserted. X-ray (Doctor A. J. Quimby)—"negative for skull; fracture of lower end of left fibula."

Treatment.—Appropriate position and plaster applied by Doctor R. E. Brennan. Liquid diet.

Examination (36 hours after admission).—Temperature, 99.8°; pulse, 86; respiration, 26; blood-pressure, 138. Patient complains of severe frontal headaches; also pain in left ankle. Reflexes negative. Fundi negative. Soft diet.

Examination at discharge (38 days after injury).—Temperature, 98.8°; pulse, 76; respiration, 22; blood-pressure, 142. No complaints except for stiffness of left ankle and in the lumbo-sacral region. Reflexes negative.

Fundi negative. Laceration of scalp healed entirely and plaster has been removed from the left ankle.

Examination (April 12, 1917—5 months after injury).—Patient complains of "light-headedness" at times; is afraid to walk up or down stairs without a cane for fear of falling as the result of dizziness. Reflexes negative. Fundi negative. No nystagmus; no Romberg.

Last Examination (September 17, 1918—22 months after injury).—Patient still complains of spells of dizziness, especially in the morning; also a fear of falling, causing her to stagger. Still uses a cane. Pupils equal and react normally. No nystagmus. No Romberg. Reflexes present and equal. Fundi negative.

Remarks.—The patient has a lawsuit pending against the owner of the automobile; it will be interesting to note if a disappearance of these annoying symptoms will occur after a satisfactory settlement at the trial; this occurs frequently in this type of conditions which may be classed as post-traumatic neuroses; it may not, however, be influenced by the successful termination of the suit, although many patients having this type of complaint following cranial injuries are usually entirely relieved after a satisfactory settlement legally has occurred—frequently within one week after the case has ended in court.

The long hospital residence of this patient was due to the fracture of the left leg and not as a result of the cranial injury; in some patients it might be said that this prolonged residence in the hospital is very beneficial to the patients in that it prevents them from returning to an active life too early and before the intracranial condition has returned to normal entirely, so that, when these patients that have been detained in the hospital on account of an injury to an extremity, such as a fracture of a bone, when they do finally leave the hospital, there is very little danger of their "overdoing" and thus tending to precipitate all kinds of "nervous" disturbances and emotional annoyances.

The importance of having these extensive scalp lacerations heal *per primam* is essential. How this patient escaped having a serious infection of the extensive scalp lacerations is very impressive and it emphasizes the great value of careful shaving of a wide area about the scalp lacerations, and then their cleansing with green soap and water and the use both of a weak solution of warm bichloride dressing (1–5000) and then either at the same time or later the free use of iodine to cleanse the lacerated tissue. Naturally, great care should be taken to ascertain first whether a fracture of the underlying bone is present, and if it should be present, then the most careful application of the solution of iodine for fear some of it might penetrate intracranially, and if the underlying dura should also be torn then the great danger of cortical irritation and the production of convulsive seizures. (I have seen this complication occur in the accident room of a hospital where iodine was freely used to "swab out" an extensive laceration of the scalp; it was afterward ascertained at operation that the major convulsion which occurred immediately after the use of iodine, had been due to the fact that some of the iodine had reached the cerebral cortex through an adjacent underlying fracture of the skull and a torn dura. Fortunately, this patient recovered and apparently no ill-effects from the medicinal cortical irritation have been permanent.)

B. *Large hematoma.*

Case 4.—Concussion; extensive hematoma; multiple contusions; dislocation of both shoulders. Recovery.

No. 49.—James. Fifty-two years. White. Married. Collector. Ireland.

Admitted June 20, 1914, Polyclinic Hospital. Referred by Doctor Alexander Lyle.

Discharged July 6, 1914—16 days after injury.

Family history negative.

Personal History.—Ten years ago, patient had yellow fever.

Present Illness.—Patient fell a distance of one story while sleeping upon a fire-escape; recovered consciousness in the ambulance.

Examination upon admission (40 minutes after injury).—Temperature, 98.2°; pulse, 104; respiration, 28; blood-pressure, 138. Well developed and nourished; no alcoholism. Semiconscious and very much confused mentally as to time and place; bleeding only from nose. Posterior dislocation of both arms at the shoulder (as confirmed later by X-ray). Abrasions, contusions and ecchymoses of back, face, especially right eye, and both extremities; sprain of right wrist. Huge hematoma over the occiput; not tender. Pupils moderately dilated but react normally. Reflexes—patellar active and equal; no ankle clonus but a suggestive right Babinski; abdominal reflexes absent. Fundi negative. No paralyses. No abdominal pain, tenderness nor dulness. Urine—not bloody.

Treatment.—Expectant palliative; as the shock was not severe both shoulder dislocations were immediately reduced. Firm, warm, wet bichloride (1–5000) dressing applied to hematoma of head.

Examination (10 hours after admission).—Temperature, 101.4°; pulse, 80; respiration, 24; blood-pressure, 160. Still semiconscious but restless. Vomited over one pint of blood; abdomen apparently negative. Hematoma over entire occiput very tense; X-ray (Doctor A. J. Quimby) did not reveal any underlying fracture of the vault and therefore merely a firm gauze bandage was applied. Pupils equal and react normally. Reflexes—patellar present and equal; no Babinski; abdominal reflexes depressed but equal. Fundi negative, except for possibly a slight dilatation of the retinal veins. Lumbar puncture—cerebrospinal fluid clear and under normal pressure (approximately 9 mm.).

Examination (48 hours after admission).—Temperature, 100.8°; pulse, 78; respiration, 24; blood-pressure, 154. Conscious but still mentally confused. Complains of pains "all over" head, shoulders and back. Hematoma possibly not so tense. Reflexes negative. Fundi negative. Soft diet.

Examination at discharge (10 days after admission).—Temperature, 98.6°; pulse, 70; respiration, 22; blood-pressure, 146. No complaints other than a general soreness and stiffness—particularly about both shoulders and back. Hematoma over occiput less extensive and not tense. Reflexes negative. Fundi negative.

Last Examination (September 17, 1981—46 months after injury).—No complaints. Reflexes negative. Fundi negative.

Remarks.—If there had been an underlying fracture at the site of the occipital hematoma in this case, it would have been most important to have kept the overlying scalp in the best possible condition at least, so that the danger of an abrasion and contusion of the scalp becoming infected will be lessened as much as possible; the application of a warm, weak, wet bichloride (1–5000) dressing seems to be very effective in keeping abrased and contused areas of the scalp from becoming infected. Only too frequently a hematoma of the scalp becomes infected from the "poor" condition of the overlying scalp and then if there is an underlying fracture of the skull, the risk of infection extending through the fracture down to the meninges is very great indeed; if the dura is intact and a purulent lepto-meningitis should not occur, there is still danger of an osteomyelitis developing in the bone adjacent to the fracture and its rather frequent complication—an extradural abscess and only too frequently a later cerebral abscess. Even in the absence of an underlying fracture of the vault (as demonstrated by X-ray) it is frequently better judgment, if there is any question of the hematoma becoming infected, to aspirate it through a "clean" area of the overlying scalp, and if necessary, to make a small incision, and thus lessen the danger of an infected hematoma allowing the infection to extend by the extracranial veins, lymphatics, etc., down into the large venous sinuses and to the meninges themselves. Only too frequently a purulent meningitis and infected sinus thrombosis occur by means of this channel of infection.

The age of these patients is an important factor in their excellent recovery; the condition upon admission is frequently such that if they are of middle age or over, there will be grave doubts whether a recovery of life is possible or not. Unless there is a serious intracranial lesion, such as an extensive cerebral laceration or a large intracranial hemorrhage, patients under sixteen years will recover as far as life is concerned even when the prognosis would seem almost hopeless. That is, the treatment and prognosis of cranial injuries in children up to sixteen years of age is radically different and the outlook always more hopeful than in similar cranial injuries in adults and particularly above middle age.

C. *Infected lacerations of the scalp.*

Case 5.—Cerebral concussion; infection of extensive laceration of scalp; severe cellulitis requiring multiple incisions and drainage. Recovery.

No. 28.—Charles. Forty-four years. White. Single. Chauffeur. Sweden.

Admitted December 12, 1914, Polyclinic Hospital. Referred by Doctor C. R. Hancock.

Discharged January 22, 1915—41 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While walking along the street, patient fell into an excavation of 6 feet, striking head against an iron pipe; unconscious upon admission to the hospital in the ambulance.

Examination upon admission (50 minutes after injury).—Temperature, 98.2°; pulse, 110; respiration, 30; blood-pressure, 124. Unconscious and in shock. Head dripping blood from a large ragged laceration over right frontal and right parietal areas—7 inches in length; dirt and hair rubbed into the tissues. No bleeding from nose, mouth or ears. No neurological examination made at this time.

Treatment.—Head shaved (unfortunately, rather carelessly), scalp laceration cleansed with green soap and then iodine swabbed into it; branches of temporal artery required ligation; wound loosely sutured and 5 rubber tissue drains inserted; firm dressing applied. The treatment of shock consisted of external warmth, quiet (morphia being ordered for any restlessness), and hot rectal saline irrigation.

Examination (6 hours after admission).—Temperature, 99.8°; pulse, 94; respiration, 22; blood-pressure, 128. Condition much better. Conscious but drowsy; orientation fair. Pupils equal and react normally. Reflexes active but equal; no ankle clonus nor Babinski; abdominal reflexes are obtained with difficulty but are apparently equal. Fundi negative. Lumbar puncture—cerebrospinal fluid clear and the pressure was normal (approximately 8 mm.); cell count was 8 per c.mm. X-ray (Doctor A. J. Quimby)—"no fracture of the skull revealed."

Treatment.—Laceration of the scalp dressed; all bleeding had ceased; wound again swabbed with iodine and a firm head bandage applied. Liquid diet.

Examination (48 hours after admission).—Temperature, 100.8°; pulse, 92; respiration, 24; blood-pressure, 142. Conscious and complains of severe pain at site of laceration, which upon dressing is reddened about the edges, indurated and distinctly tender; boggy edema to a distance of two inches beyond the lacerated tissue. Drains removed and wound again swabbed out with iodine; drains reinserted; large wet bichloride (1–5000) dressing applied. Pupils equal and react normally. Reflexes negative. Fundi negative.

Treatment.—Nothing but liquids; force water; vigorous catharsis. Daily dressings. Quiet.

Examination (4 days after admission).—Temperature, 105.4°; pulse, 120; respiration, 32; blood-pressure, 164. Irrational and extremely restless, requiring restraint. Laceration discharging thin watery secretion. Laboratory report (Doctor Jeffries)—"streptococci." Edema of entire scalp extending forward to both orbits and closing both eyes and also downward about ears and backward into the neck. Wound reopened, loosely packed with wet alcohol gauze after being swabbed with iodine; without an anesthetic, multiple radiating incisions—7 in all—made over the parietal and occipital regions adjacent to the laceration and numerous rubber tissue drains inserted; incisions not sutured. No frank pus obtained by the incisions but much watery edema escaped, lessening the tension of the scalp tissues. Lumbar puncture—clear cerebrospinal fluid under normal pressure (approximately 8 mm.); cell count was 8 cells per c.mm. Reflexes negative. Fundi—slight dilatation of retinal veins but no definite edema of the optic disk margins. No stiffness of neck; no Kernig obtained.

Treatment.—Liquid diet; force water; vigorous catharsis.

Examination (5 days after admission—18 hours after incising the scalp).—Temperature, 101.8°; pulse, 102; respiration, 26; blood-pressure,

144. Patient less irrational and not so restless. Condition better in every way. Edema of scalp markedly lessened and both eyes can now be opened. No definite rigidity of neck; no Kernig. Reflexes negative. Fundi—dilatation of retinal veins still persists but otherwise negative.

Treatment continued as above.

Examination (10 days after admission).—Temperature, 99.2°; pulse, 84; respiration, 22; blood-pressure, 138. Perfectly rational and no complaints except for general soreness of head. Multiple incisions still discharging a thin watery pus but no marked tenderness or redness present. Original laceration of scalp beginning to granulate. No edema of scalp and eyes can be widely opened. Pupils equal and react normally. Reflexes negative. Fundi negative. Soft diet.

Examination at discharge (41 days after admission).—Temperature, 98.8°; pulse, 76; respiration, 20; blood-pressure, 132. No complaints except for a general stiffness and numbness of scalp—undoubtedly due to the multiple incisions. The original laceration of scalp is practically healed except for a small granulating area at its posterior angle. Reflexes negative. Fundi negative.

Examination (November 23, 1915—11 months after injury).—No complaints except for slight headache at times. Reflexes negative. Fundi negative.

Last Examination (March 5, 1918—39 months after injury).—No complaints. Reflexes negative. Fundi negative. General contracture of scalp due to scar tissue of incision.

Remarks.—This was the most severe condition of infection and cellulitis of the scalp that I had ever seen; for a time it seemed that nothing would check the rapid progress of the infection, and yet at no time did the patient's neck become rigid nor did the cell count of the cerebrospinal fluid increase beyond 8 cells per c.mm. It would appear that the extensive multiple incisions of the scalp had in this case afforded such excellent drainage that the infective process was "headed off"; the application of the continuous head bath of warm, weak bichloride (1–5000) dressing was undoubtedly a big factor in the excellent result.

This patient had a very narrow escape; the danger of a resulting meningitis or an infected sinus thrombosis was very great indeed. If a fracture of the underlying skull had been present, then it is very doubtful if a purulent meningitis could have been avoided. In treating infections of the scalp of this character and also the more typical form of purulent cellulitis, free open drainage is essential, and then next in importance in the local treatment is the continuous application of warm, weak, "wet" bichloride (1–5000) dressings—in fact, a sort of head bath; if a bichloride solution is not thought advisable, then merely warm normal saline solution may be used—similar to the warm "soaks" for cellulitis of the arm or leg—a continuous warm bath for the part affected. The results obtained by this method of treatment have been most encouraging.

D. *Alcoholism.*

Case 6.—Severe cerebral concussion; alcoholism. Recovery.

No. 36.—Benjamin. Fifty years. Married. White. Bookbinder. Ireland.

Admitted June 14, 1914, Polyclinic Hospital. Referred by Doctor John A. Bodine.

Discharged June 19, 1914—5 days after injury.

Family history negative.

Personal history negative, except for alcoholic excesses.

Present Illness.—Patient was found at bottom of stairway; said to have fallen one flight after drinking heavily; profound unconsciousness. Brought to the hospital in the ambulance.

Examination upon admission (about one hour after injury).—Temperature, 98°; pulse, 88; respiration, 16; blood-pressure, 146. Semiconscious—not easily aroused but reacted to ammonia fumes; heavy alcoholic breath; deep stertorous breathing but regular. Bleeding from nose but no cerebrospinal fluid observed. Superficial abrasion of right temporal region; marked ecchymosis of right eye; abrasion of right knee and left wrist. Pupils slightly enlarged but react to light sluggishly. Reflexes—knee-jerks absent; ankle jerks absent, elbow and wrist jerks not elicited; no ankle clonus; no Babinski; abdominal reflexes not obtained. Fundi negative. Lumbar puncture—cerebrospinal fluid clear and not under pressure (approximately 8 mm.).

Treatment.—Expectant palliative treatment.

Examination (7 hours after admission).—Temperature, 99.4°; pulse, 74; respiration, 20; blood-pressure, 140. Patient is still unconscious. Respiration deep but regular. Pupils equal and react normally. Reflexes elicited as at preceding examination. Fundi negative. X-ray (Doctor A. J. Quimby)—"negative."

Examination (18 hours after admission).—Temperature, 99.2°; pulse, 78; respiration, 22; blood-pressure, 148. Patient regaining consciousness; marked disorientation as to time and place. Complains of piercing occipital headache and general pains "all over body." Pupils equal and react actively. Reflexes—knee-jerks difficult to elicit but equal; no ankle clonus nor Babinski; abdominal reflexes not obtained. Fundi negative.

Treatment.—Prophylactic alcoholic treatment; active catharsis.

Examination at discharge (5 days after admission).—Temperature, 98.4°; pulse, 76; respiration, 20; blood-pressure, 144. Complains of mild headache; otherwise well. Pupils equal and react normally. Reflexes present and equal. Abdominal reflexes depressed but equal. Fundi negative. Patient signed the pledge regarding alcoholic abstention.

Examination (July 4, 1914—20 days after injury).—Patient was seen drunk holding to a lamp-post. A superficial examination upon a doorstep revealed nothing abnormal except depressed reflexes undoubtedly due to the alcohol.

Examination (October 12, 1914—4 months after injury).—No complaints. Reflexes negative. Fundi negative.

Last Examination (May 12, 1918).—No complaints. Patient has deteriorated very much from alcohol. Pupils equal but react sluggishly. Reflexes present but uniformly depressed. Fundi negative, except for a retinal suffusion. Urine contains much albumen and many granular casts. Medical treatment advised.

Remarks.—This patient has deteriorated so much from alcoholism since the former injury that it is very doubtful if he could withstand successfully the effects of a similar injury now; delirium tremens would occur in all probability, and the patient would be most fortunate if he escaped with his life. Whether the cranial injury with the resulting so-called "concussion" contributed in any way to the patient's greater susceptibility to alcohol and the marked mental and emotional deterioration cannot be stated with any degree of accuracy; it would seem, however, that such a cranial injury could at least do him no good, and possibly permitted him to deteriorate much more rapidly than ordinarily or if the injury had not occurred.

The danger of delirium tremens developing in patients of this character is very great indeed; it is naturally not so great when the patient is confined to bed only for several days, but this factor of alcoholism must always be considered in the treatment of patients having head injuries—no matter how apparently trivial the injury may appear to be.

E. *Existing mental derangement.*

Case 7.—Severe cerebral concussion occurring in a patient mentally deranged; paranoia.

No. 708.—Camille. Forty years. White. Single. Playwright. U. S.
Admitted October 13, 1916, Polyclinic Hospital.
Discharged November 26, 1916—43 days after injury.

Family history negative.

Past History.—One week before the injury, relatives had filled out the necessary papers and physicians had advised the patient to be committed to an institution for the mentally deranged—the diagnosis being paranoia.

Present Illness.—While crossing the street, patient was struck by an automobile and knocked down; quickly recovered consciousness, and was brought in the ambulance to the hospital in a semiconscious condition.

Examination upon admission (one hour after injury).—Temperature, 98.4°; pulse, 80; respiration, 26; blood-pressure, 138. Extremely irrational—almost maniacal—requiring restraint. Insists that she is the original vampire and therefore called "Camille." She says that the attendants show her no longer the respect due her—"a second Cleopatra." A well-nourished woman having a laceration, 2 inches long, over left frontal area and bruises over entire body. Pupils equal and react normally. Reflexes—knee-jerks exaggerated equally; double ankle clonus, and suggestive right Babinski; abdominal reflexes absent. Fundi—slight dilatation of retinal veins; otherwise negative.

Treatment.—Expectant palliative; usual treatment of scalp laceration.

Examination (48 hours after admission).—Temperature, 99.8°; pulse, 86; respiration, 24; blood-pressure, 142. No complaints. Patient less noisy but disoriented; continues to rave about "Cleopatra" and the "saints." Reflexes—active but good; no ankle clonus nor Babinski; abdominal reflexes active and equal. Fundi negative. Lumbar puncture—cerebrospinal fluid clear and under normal pressure (approximately 9 mm.). X-ray (Doctor W. H. Stewart)—"negative for fracture of the skull."

Treatment.—Restraint as required. Soft diet.

Examination (8 days after admission).—Temperature, 98.8°; pulse, 82;

respiration, 24; blood-pressure, 140. Patient has been normally quiet di ing the past 10 days. Pleasant, cheerful and has not mentioned her "i portance" and her "power." Laceration of scalp has healed nicely. I flexes negative. Fundi negative.

Examination at discharge (13 days after admission).—Temperatu 98.8°; pulse, 78; respiration, 22; blood-pressure, 138. No complaints "always feels fine." Patient has refused to eat all food except liqui saying "it's all poisoned." Other than this, patient apparently norm except for egotistical attitude toward the writing of short stories—"I a an unusual novelist—in fact, a second Jane Austen." Reflexes negati Fundi negative.

Treatment.—Patient was given into the custody of her relatives, w removed her to the South for the winter.

Examination (August 1, 1917—10 months after the injury).—Acco ing to the relatives and also the patient she "calmed down" so much aft the accident that institutional care is unnecessary and it has been possil for her to live quietly in the family again; her responsibilities are negligib Reflexes active but equal; no ankle clonus nor Babinski. Fundi negative.

Last Examination (August 10, 1918—22 months after injury).—Patie is now living in Greenwich Village, New York City, among "artists" conducting a tea shop; is able to earn her living and though "eccentric still she is considered normal for that community. Reflexes negati Fundi negative.

Remarks.—The rest in the hospital during the 6 weeks following t injury and then the vacation of the patient to the South where she w removed from cares, worries and emotional stress—these two factors u doubtedly improved the patient's mental condition very much so that s was really better one year after the injury than before the injury. T subsequent history will be very interesting, although it seems that the he injury has only been an incident in this patient's condition.

F. *Pneumonia, senility and decubitus.*

CASE 8.—Cerebral concussion; pneumonia, decubitus, and senili Death. Autopsy.

No. 1006.—Margaret. Sixty-six years. White. M Ireland.

contracted equally and reacted to light normally. Reflexes—negative, except for a suggestive left Babinski. Fundi negative.

Treatment.—Expectant palliative and vigorous shock measures.

Examination (12 hours after admission).—Temperature, 99°; pulse, 86; respiration, 20; blood-pressure, 146. Patient now semiconscious and very restless, requiring morphia and restraint. Pupils equal and react normally. Reflexes active and equal. Fundi negative. Lumbar puncture—cerebrospinal fluid clear and under normal pressure (approximately 7 mm.). X-ray (Doctor G. W. Welton)—"no fracture revealed."

Treatment.—Expectant palliative; liquid diet.

Examination (4 days after admission).—Temperature, 99.2°; pulse, 82; respiration, 26; blood-pressure, 152. Patient now conscious but drowsy; confused mentally and badly orientated as to time, place and personality (relatives, however, state that she has been feeble-minded and childish; during the past year she has strayed away from home and was lost when the accident occurred). Patient continuously says she is going home and must be restrained in bed in order to prevent her from walking into the corridors of the hospital. She coughs frequently and auscultation reveals many coarse and sibilant râles throughout both lungs, particularly their lower halves. Numerous ecchymotic areas have appeared over body so that owing to general soreness and tenderness patient cannot be moved from side to side without causing her pain. Pupils equal and react normally. Reflexes—active but equal; no ankle clonus nor Babinski present. Fundi negative.

Treatment.—Owing to the great weakness of the patient and her inability to do anything herself (even swallowing being a great effort for her), great care is taken in feeding her liquids and soft diet; she is turned frequently in bed from side to side and a low back rest is used in order to lessen the danger of pulmonary complications.

Examination (10 days after admission).—Temperature, 101.4°; pulse, 90; respiration, 28; blood-pressure, 152. Patient conscious but irrational. In spite of great care, bed sores have developed on each side of the sacrum (apparently air cushions were not sufficient and an air or water mattress would have been advisable). Patient had been gotten out of bed in a chair on the previous day, but she had to be quickly returned to bed owing to her great general weakness. Numerous moist râles throughout both bases of lungs. Reflexes negative. Fundi negative.

Examination (15 days after admission).—Temperature, 103.2; pulse, 94; respiration, 34; blood-pressure, 134. Patient irrational and extremely weak. Dulness over right lower half of chest with a marked diminution of respiration over this area; numerous coarse râles.

Treatment.—For several days, the treatment has been directed toward the chest condition—slight elevation in bed, frequent turning and small repeated doses of atropine (grains 1/100 every 2–4 hours). The patient, however, is so weakened that she does not react as she should.

Examination (4 hours before death—20 days after admission).—Temperature, 106.4; pulse, 138; respiration, 44; blood-pressure, 110. Patient unconscious; marked cyanosis with shallow irregular respirations. Dulness over both bases and lower halves of lungs.

Autopsy.—Head: no fracture of skull ascertained; no intracranial hemorrhage, merely a wet edematous brain not under tension. Chest: double hypostatic pneumonia. Abdomen negative.

Remarks.—At the time of the death of this patient, it was my opinion that it could possibly have been avoided if the patient had been "gotten out of bed" and into a wheel chair upon about the sixth day after the injury; it is so important for elderly patients having cranial injuries not to be confined to bed for a period longer than one week, and the longer they are confined in bed the greater is the risk of the complication of pneumonia, decubitus and the rapid mental deterioration; this latter complication is frequently sufficient in itself to hasten the exitus, and especially when associated with one or more bed-sores, as it seems that both the mental and physical resistance reaches such a low point that the body-cells simply refuse to functionate.

The autopsy findings of a mild condition of cerebral edema and not under tension are almost always found in patients following a prolonged illness of this character, and especially if the patient is at all alcoholic.

I. Doubtful "Fractures of the Skull"

Cranial injuries of sufficient severity to produce a fracture of the skull frequently damage the underlying delicate tissues of the brain and the intracranial vessels; for this reason, these injuries became popularly known as "fractures of the skull" rather than the important designation of "brain injuries"—in fact, these two terms were almost synonymous and used interchangeably. Within recent years it has been surprising to ascertain that "fractures of the skull" often occur without an injury to the underlying brain or a marked increase of the intracranial pressure, whereas cranial injuries without a fracture of the skull very frequently cause such a severe damage to the brain from the resulting increased intracranial pressure of hemorrhage or edema that an early death will occur unless this pressure is immediately relieved and, unfortunately, at times even with an operation. Careful autopsy records of these patients indicate the great frequency of death due to cranial injuries in the absence of any fracture of the skull—the most common direct cause of the death being a high intracranial pressure due to cerebral edema and eventually producing a medullary edema; the initial shock following the cranial injury is also a frequent factor, and then the condition of intracranial hemorrhage; the fracture of the skull, however, is comparatively unimportant.

The following case-histories are selected to illustrate the relative unimportance of the presence or not of a fracture of the skull (naturally, depressed fractures of the skull are excluded), and yet every modern means of examination should be employed in order that a complete clinical picture of the condition should be elicited. If there are not present the signs of severe shock or of a marked increase of the intracranial pressure, then the condition of the patient is in no way aggravated by repeated neurological examinations, röntgenograms, the use of the ophthalmoscope and of the spinal mercurial manometer; but to make these examinations while the patient is in a severe condition of initial shock or to delay the operation of cranial decompression—even "over-night"—when the intracranial pressure is high, merely because an X-ray picture has not been obtained, is a mistake of the gravest concern and has been the cause of many deaths. The presence or not of a fracture of the skull and its location is of no real importance in either the diagnosis of the intracranial condition or in the treatment, and the appropriate treatment must never be delayed merely to ascertain its presence. If the line of fracture should open into the middle ear and the tympanic membrane be ruptured, or into the nasal or pharyngeal cavities, naturally the risk of infection and possible meningitis would be greater, and yet this is a complication which not only rarely occurs if aural irrigation is avoided and no "meddling" occurs, but the condition itself is usually easily elicited by inspection alone—or at most by an otoscopic and rhinoscopic examination; a positive röntgenogram would in no way change the treatment, unless it disclosed a depressed fracture of the vault and it is for this latter reason chiefly that röntgenograms are of importance in all patients having cranial injuries.

RECENT DOUBTFUL FRACTURES OF THE SKULL; NO MARKED SIGNS OF INCREASED INTRACRANIAL PRESSURE; NO OPERATION

CASE 9.—Doubtful fracture of the skull; mild signs of an increased intracranial pressure; fracture of right humerus, right ankle and right scapula. No operation. Recovery.

No. 65.—Andrew. Fifty-two years. White. Single. Salesman. Germany. Admitted September 2, 1914, Polyclinic Hospital. Referred by Doctor W. S. Bainbridge.

Discharged September 30, 1914—28 days after injury.

Family history negative.

Personal history negative.

Present Illness.—Patient was struck by a trolley-car; unconscious for several minutes; brought to the hospital in the ambulance.

Examination upon admission (1 hour after injury).—Temperature, 98.8°; pulse, 96; respiration, 30; blood-pressure, 146. Rather obese but robust. Conscious for short periods but badly oriented; no alcoholism. Bleeding slowly from right ear; no cerebrospinal fluid observed; no mastoid ecchymosis at this examination. Hematoma and abrasion of right cheek and face; ecchymosis of both eyes, particularly right; right shoulder swollen and ecchymosed. Pupils moderately dilated, equal, and react normally. Reflexes negative, except for a suggestive right Babinski. Fundi negative.

Treatment.—Expectant palliative treatment and for shock.

Examination (48 hours after admission).—Temperature, 99.8°; pulse, 84; respiration, 22; blood-pressure, 140. Conscious; oriented, but remembers nothing of the day of the accident. Complains of dull throbbing headache. Slight weakness of right side of face (peripheral type of facial paralysis). Bleeding of right ear has ceased; slight mastoid discoloration and slightly tender; otoscopic examination reveals a small laceration of posterior lower quadrant of right tympanic membrane. Pupils equal and react normally. Reflexes active but equal; no ankle clonus nor Babinski. Fundi: slight dilatation of retinal veins and a hazy edema along the nasal margins of the optic disks—right being possibly greater than left. Lumbar puncture: cerebrospinal fluid straw-colored (laboratory reports "numerous red blood-corpuscles"); under a pressure of approximately 12 mm. X-ray (Doctor A. J. Quimby)—"no fracture of skull revealed; fracture of the greater tuberosity of the right humerus and a transverse fracture of the right scapula; fracture of the internal malleolus of the right ankle."

Treatment.—Expectant palliative; appropriate splints applied to the right shoulder and right ankle.

Examination (10 days after admission).—Temperature, 102.2°; pulse, 86; respiration, 24; blood-pressure, 142. Complains of throbbing pain in right cheek at the site of the hematoma, which has become "hot and tender." (Hot, wet, gauze dressing of normal saline continuously used and the hematoma did not break down into frank pus; after 24 hours, its size lessened as well as "the pain and tenderness.") Reflexes active but equal. Fundi negative.

Examination at discharge (28 days after admission).—Temperature, 99°; pulse, 78; respiration, 24; blood-pressure, 144. No complaints except for limitation of movement and soreness of right shoulder and right ankle; occasional "light-headed" spells. No weakness of right side of face elicited by the special tests. Reflexes negative. Fundi negative.

Examination (May 12, 1915—8 months after injury).—Complains of headache, particularly in the morning just after awakening, and of periods of dizziness if he should stoop suddenly; also being easily fatigued—becomes tired in the middle of the day when he has his afternoon's work ahead of him. (These complaints, however, all disappeared within a month after the satisfactory settlement of his suit against the traction company.) Reflexes active but equal; no ankle clonus nor Babinski. Fundi negative.

Examination (March 26, 1917—31 months after injury).—No complaints whatever; rather euphoric—alcoholism. Reflexes negative although active. Fundi negative.

Last Examination (September 17, 1918—48 months after admission).—No complaints; "feels fine." Alcoholism has become more pronounced. Reflexes active but equal; no Babinski. Fundi negative.

Remarks.—The mere fact of blood escaping from the right auditory canal might easily have been considered as undoubtedly due to a fracture of the base of the skull extending through the right ear, and yet if no cerebrospinal fluid is observed in the discharge and the otoscopic examination does not reveal a laceration of the homolateral tympanic membrane, and the X-ray is negative, then it cannot be stated with certainty that there is present a fracture of the skull. In this patient, however, although there was only blood observed in the discharge from the ear, and the X-ray was negative (as they usually are in fractures of the base alone), the otoscopic examination did reveal a laceration of the posterior portion of the right tympanic membrane, and it is rare for this complication to occur unless the adjacent bone has been fractured; it is possible, however, for a small object to have torn the membrane by penetrating the external auditory canal and yet there are usually signs of this external injury in the auditory canal itself, and it is a very rare occurrence. It is my opinion that this patient did have a fracture of the base of the skull in the region of the petrous portion of the right temporal bone and yet it is impossible to demonstrate it clinically—other than the fact that the right tympanic membrane has been ruptured.

The mild signs of an increased intracranial pressure, as revealed by the ophthalmoscope and at lumbar puncture, were not of sufficient degree to warrant the operation of cranial decompression, and these are the patients who make excellent recoveries with the expectant palliative treatment alone by the natural absorption of the small amount of intracranial hemorrhage (if present) and the resulting mild cerebral edema. In this patient, however, the absorption of the cerebral edema has been complicated and possibly delayed by the factor of alcoholism; whether this increased indulgence of alcohol is due to an emotional instability resulting from the cranial injury or is a natural sequence of his former life and habits, cannot be asserted with any degree of accuracy; the cranial injury, however, surely could not have been of benefit to either his mental or emotional status.

If the hematoma of the right side of the face had become infected to the point of pus formation and if there was a fracture of the base of the skull adjacent to the right ear, the danger of this infection extending to the meninges through the fracture of the skull would have been very great indeed; if there is any question of pus formation in the vicinity of the fracture of the skull, the hematoma and abscess (if formed) should be incised and free drainage afforded in order to avoid any serious complications.

It is very easy to make the diagnosis of "depressed fracture of the skull" when palpation alone is used, especially in the presence of a hematoma or a marked edema of the overlying scalp, if the adjacent pericranium has been torn, because it gives the sensation of a depressed edge of bone; röntgenograms are most valuable for this differentiation, but if there is still doubt then a small exploratory incision of the overlying scalp would be advisable—the risk being small, if any, and if there should be a depressed fracture of the vault now is the time to know it, so that it can either be elevated or removed before definite signs of its presence intracranially are exhibited. The weakness of right half of face (peripheral in type), associated with a definite impairment of hearing of the right ear (a lesion of the middle ear), and with an ecchymosis of the right mastoid area, would indicate a lesion of the facial nerve in its bony aqueduct about the middle ear—either a fracture of the adjacent bone or merely a temporary edema of the nerve itself within its bony canal and thus producing a temporary compression paresis of the ipsolateral right half of the face. The right tympanic membrane being intact and of normal appearance would tend to exclude a fracture of the adjacent bones and yet the impairment of hearing, being referable to the middle ear, would point to a possible fracture in the petrous portion of the right temporal bone.

CASE 10.—Doubtful fracture of the skull; no signs of an increased intracranial pressure. No operation. Recovery.

No. 87.—Lilly. Forty-eight years. Colored. Married. Housework. U. S.
Admitted February 28, 1915, Polyclinic Hospital.
Discharged March 23, 1915—27 days after injury.
Family history negative.
Personal history negative.

Present Illness.—Three days before admission, patient was struck by a large piece of ceiling falling upon the back of her head and neck; says she was unconscious about 15 minutes and was aroused by friends. Had severe headache and pain in neck and back during next 2 days; was sent in a cab to the hospital.

Examination upon admission (10 hours after injury).—Temperature, 99.4°; pulse, 84; respiration, 24; blood-pressure, 168. Well developed and well nourished negress; heart and abdomen negative. Few fine moist râles over apices of both lungs. Conscious but drowsy. Complains of severe occipital headache and dizziness; pain in her eyes and throughout her neck and back; slight stiffness of neck. Slight contusion over occiput but no other external local marks of injury ascertained. Some clotted blood in left auditory meatus; definite ecchymosis behind left ear over mastoid. Reflexes active, the right knee-jerks being possibly greater than the left; no Babinski. Pupils equal and react normally. Fundi negative. Lumbar puncture—cerebrospinal fluid clear and under normal pressure (approximately 9 mm.). X-ray (Doctor A. J. Quimby)—"negative."

Treatment.—Expectant palliative.

Examination (6 days after admission).—Temperature, 101.2°; pulse, 86; respiration, 22; blood-pressure, 156. Conscious; still complains of soreness "all over" and particularly at back of head. No real rigidity of neck; no Kernig. Otoscopic examination reveals small laceration of the left tympanic membrane—in its lower posterior quadrant. Left mastoid ecchymosis more marked; some tenderness upon deep palpation. Pupils equal and react normally. Reflexes—knee-jerks sluggish but equal; no ankle clonus, Babinski, Oppenheim, Gordon. Fundi (ophthalmoscopic examination by Doctor J. A. Kearney)—"external condition normal; both eyes are moderately myopic and pigmented retinæ peculiar to negroes. Optic disks—media clear; no dilatation of veins of fundus; arteries and veins in normal relation as to color; disks slightly pale, edges clearly cut all around." Lumbar puncture attempted but not successful owing to the rolls of fat in the lumbar region; an infective meningitis was feared.

Treatment.—Expectant palliative continued.

Examination (12 days after admission).—Temperature, 99.4°; pulse, 80; respiration, 24; blood-pressure, 154. Still complains of headache, soreness "all over" and stiffness of neck. (X-ray of cervical region of spine r [illegible] tive.) Left mastoid ecchymosis has disappeared. Reflexes active but e [illegible] Fundi negative. No Kernig nor real rigidity.

[illegible] *mination* at discharge (24 days after admission).—Temperature, [illegible] lse, 76; respiration, 22; blood-pressure, 156. Much better but still [illegible] s of headache, dizziness and stiffness of neck. Reflexes negative. F [illegible] tive.

[illegible] *ion* (January 10, 1916—11 months after injury, and just before her case was to be tried in court).—Same complaints persist: headache, "light-headed" spells in the morning and when stooping and inability to turn neck on account of pain. Pupils equal and react normally. Reflexes active but equal. Fundi negative. Hearing slightly impaired in left ear—Weber's test being positive (bone conduction being better than air conduction) and therefore middle ear impairment.

Last Examination (September 17, 1918—42 months after injury and following an unsatisfactory case (to her) in court).—"Never just as well as before injury." General complaints—nothing of any great severity or annoyance, however. Reflexes negative. Fundi negative.

Remarks.—On account of the inability to perform a second lumbar puncture on the sixth day after admission and the patient having a rise of temperature with pain and stiffness of the neck, this caused a fear that a meningitis might be occurring in this patient—possibly through an infection of the left middle ear, the left tympanic membrane having been lacerated; fortunately this condition did not occur and the inability to perform the lumbar puncture was due undoubtedly to its being difficult technically—the patient being rather obese.

Although the X-ray did not reveal a fracture of the skull in this patient, yet the presence of a laceration of the left tympanic membrane is very indicative that a fracture of the skull, at least of the left petrous bone, did exist; the necessity for possibly more care and frequent examinations would naturally be more strongly emphasized—the physician realizing that the cranial injury had been a sufficient force to cause a fracture of the skull and thus the possible danger of an intracranial lesion must be remembered; besides the ever-present danger of a purulent meningitis and its complications resulting from an infection through the line of fracture, especially of the ear, nares and pharynx.

It is interesting to note that in this patient, too, the complaints of headache, dizziness and stiffness of the neck, all became much less and practically disappeared after the termination of the patient's lawsuit—even though the settlement was not satisfactory to her; the mere relief, both mental and emotional that "the case is now over and I am glad of it" was sufficient to cause an almost immediate improvement in this patient's condition. I believe that a large percentage of post-traumatic neuroses are based upon the question of a satisfactory lawsuit or not and that when a satisfactory settlement has occurred, these patients improve remarkably quickly.

An otoscopic examination should be made a routine procedure upon all patients having cranial injuries; in this manner, it can be accurately ascertained whether a bloody discharge from either auditory canal is due simply to an abrasion of the canal wall itself or whether there is a laceration of the tympanic membrane, due usually in cranial injuries to a fracture extending through the petrous portion of the temporal bone; naturally the discharge of cerebrospinal fluid from the external auditory canal presupposes the presence of a fracture of the skull opening into the middle ear and a rupture of its tympanic membrane. It occasionally happens that bleeding occurs in the middle ear following a cranial injury and yet the tympanic membrane is not ruptured, and therefore the otoscope reveals a tense bluish tympanic membrane; in some patients it is advisable to perform a paracentesis and thus permit the blood in the middle ear to escape in the hope that the hearing will not be impaired so much as if this clotted blood should be permitted to become organized about the tympanic ossicles. Very frequently in these patients, the impairment of hearing does not persist longer than several months, and it has been very interesting to observe a gradual increase of the auditory acuity and the change of bone conduction from being greater than air conduction to that of air conduction being greater than bone conduction.

CASE 11.—Doubtful fracture of the skull; mild signs of an increased intracranial pressure; fracture of the femur. No operation; repeated lumbar punctures. Recovery.

No. 248.—Harry. Twenty-eight years. White. Married. Painter. Poland.

Admitted May 3, 1915, Polyclinic Hospital.

Discharged June 7, 1915—29 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While painting patient is said to have fallen from a height of 7 stories; profound unconsciousness. Brought to the hospital in the ambulance.

Examination upon admission (35 minutes after injury).—Temperature, 97.6°; pulse, 142; respiration, 46; blood-pressure, 96. Extreme shock made it obligatory to avoid tiring examinations. While the vigorous treatment of shock was being instituted, inspection revealed ecchymoses of both orbits and a large hematoma over right frontal area which upon palpation was very suggestive of a depression of the underlying bone. Bleeding from nose and mouth but not from the ears; no cerebrospinal fluid observed. Complete fracture of lower third of right femur, for which a temporary box splint was used. Pupils widely dilated and reacted poorly to electric light. Reflexes not examined. Fundi negative.

Treatment.—Most active treatment for shock: external warmth, hot rectal black coffee and saline enemata; lowering of head and shoulders. (The cranial condition was not considered in detail and it should not be permitted to interfere or delay the treatment of the shock, which demands the immediate and the entire attention as being all-important.)

Examination (6 hours after admission).—Temperature, 98°; pulse, 126; respiration, 34; blood-pressure, 110. Patient is responding to the shock treatment—body becoming warmer, pulse stronger and more regular; unconsciousness is not so profound and patient moves his arms at intervals. Both eyes are closed by edema and very "black and blue." Pupils could not be examined, nor the fundi.

Treatment.—Expectant palliative.

Examination (24 hours after admission).—Temperature, 100.2°; pulse, 108; respiration, 28; blood-pressure, 126. Conscious at intervals but mentally irrational most of the time; very restless, requiring restraint. Hematoma of right frontal area very tense and giving the distinct impression, upon careful palpation, of a depressed fracture of the underlying bone. No mastoid ecchymoses. Pupils and fundi could not be examined on account of the extensive orbital ecchymoses. Reflexes of left leg very sluggish; no ankle clonus nor Babinski; reflexes of the arms were negative. Lumbar puncture—cerebrospinal fluid blood-tinged (laboratory report—"numerous red blood-cells") and under normal pressure (approximately 8 mm.). X-ray (Doctor A. J. Quimby)—"no fracture revealed (the head being taken at 5 different angles").

Treatment.—Expectant palliative; Doctor R. E. Brennan applied traction-splint to the fractured right femur.

Examination (6 days after admission).—Temperature, 99.8°; pulse, 92; respiration, 26; blood-pressure, 132. Conscious but still irrational and wants to go home. Complains of severe general headache and pain in right thigh. Hematoma of right frontal area less tense and possibly not so large. Pupils equal and react normally. Reflexes—patellar active, left more than right; no ankle clonus nor Babinski. Fundi negative. Lumbar puncture—cerebrospinal fluid straw-colored (laboratory report—"red blood-cells") and under a pressure of approximately 12 mm.

Treatment.—Owing to the constant severe headache and the increased pressure of approximately 12 mm. of the cerebrospinal fluid at lumbar puncture, it was thought advisable to withdraw 15–20 c.c. of cerebrospinal fluid daily in order to lower this mild intracranial pressure and thereby lessen the headache; a lumbar puncture was thus performed daily for 4 days with surprising and almost immediate relief and a marked general improvement; the cerebrospinal fluid appeared clear in the last 2 days of its withdrawal and the pressure on the fourth day was only approximately 9 mm. of mercury.

Examination at discharge (29 days after admission).—Temperature, 99°; pulse, 84; respiration, 26; blood-pressure, 136. No cranial complaints. Right thigh is still in a cast. No signs of the former hematoma of the scalp. Pupils equal and react normally. Reflexes active but equal; no ankle clonus nor Babinski. Fundi negative.

Examination (November 10, 1913—6 months after injury).—No cranial complaints except a "throbbing and buzzing" in the head after severe physical exertion or while stooping at a height of several stories. Right leg in excellent condition; no apparent shortening (rough measurement). Reflexes active and equal. Fundi negative.

Last Examination (July 10, 1918—34 months after injury).—No complaints. Reflexes active and equal. Fundi negative.

Remarks.—The severity of the shock was so great and extreme in this patient that it was very doubtful whether a recovery of life was possible; it is so important in patients of this extreme character to avoid all unnecessary examinations and disturbance of the patient, and that the treatment should be directed toward overcoming and lessening the condition of shock and thus improving the patient's general condition. External warmth to the entire body is possibly the most valuable procedure in combatting shock while absolute quiet and hot black coffee and saline enemata are also of great value; if the patient is at all restless, small repeated doses of morphia hypodermically should be administered.

It was interesting to observe in this patient that during the period of severe initial shock, there were no signs of an increased intracranial pressure; this is easily explained because in severe shock of this degree the pressure of the blood is very much diminished—as low as 96 as in this patient, and naturally it is not possible for a marked rise of the intracranial pressure to occur (unless immediately following the cranial injury a large intracranial hemorrhage had resulted from a tear of an unusually large cerebral or intracranial blood-vessel—much bleeding occurring before the shock had become pronounced). Then as in this patient when the blood-pressure increased as the result of the lessening of the shock, it is only then that the signs of an increased intracranial pressure begin to appear, and as the blood-pressure increases just so much more does the intracranial pressure rise until the signs of an increased intracranial pressure overshadow all the signs of the former condition of shock. In this patient, repeated lumbar punctures daily were sufficient to lessen this increased intracranial pressure and thus to "tide over" the patient until the increased pressure of free hemorrhage and cerebral edema could be "taken care of" and absorbed by natural means; in this manner the expectant palliative treatment of brain injuries in selected patients can be very much aided by the careful use of repeated lumbar punctures.

Repeated lumbar punctures in certain selected cases of brain injuries having but a mild increase of the intracranial pressure but associated with extreme headache, are of distinct and definite value if properly performed; the cerebrospinal fluid should not be allowed to escape rapidly from the needle and great care should be used in not removing too much of the fluid at any one time for fear of producing medullary disturbances and particularly a rapid lowering of the pulse- and respiration-rate due to a mild constriction of the medulla in the foramen magnum; with care, however, the danger of this complication is practically nil, and especially in those patients who have not marked signs of a high increase of intracranial pressure as revealed by the ophthalmoscope and especially by means of the spinal mercurial manometer; in these latter patients it would be distinctly dangerous to remove a large amount of cerebrospinal fluid (over 20 c.c.), so that repeated lumbar punctures should only be used therapeutically in cases where the increased intracranial pressure is mild. The most satisfactory cases are those having severe headache, and yet the increased intracranial pressure is slight; these are the ones in whom an immediate cessation of the headache occurs following the withdrawal of 10–20 c.c. of fluid, and the headache may remain absent for a period of 12 hours and even longer; usually, however, the headache returns in these patients within a period of 24 hours.

The indication for repeated lumbar punctures would be possibly greater in those patients in whom the headache and other symptoms are severe and yet the increased intracranial pressure is not high and the cerebrospinal fluid is more than merely blood-tinged; in these patients not only would there be a lessening (even though temporarily) of the increased intracranial pressure, but the lumbar puncture would be a means of withdrawing and thus draining a certain amount of free subdural and subarachnoid hemorrhage so that there would be less new tissue formation, adhesions, etc., after the absorption, as much as possible, of this free blood had taken place. The patient described above is an excellent illustration of the benefits that can be obtained by frequently repeated lumbar punctures, and it seems that in some borderline cases, that this method of repeated lumbar punctures may be the means of avoiding a later cranial decompression and drainage; however, no patient having marked signs of increased intracranial pressure and thus requiring the cranial operation of subtemporal decompression and drainage should be permitted to run the great risk of both life and future normality in order to attempt the treatment of the condition by merely lumbar punctures —a method of treatment for mild cases only, and possibly to be included in what is considered the expectant palliative treatment of brain injuries.

Fractures of the Vault of the Skull

Fractures of the vault of the skull and limited to the vault alone are usually the result of an overlying "direct" local injury in contrast to the fractures of the base of the skull resulting from an "indirect" injury; it is not common, however, for linear fractures of the vault to remain limited to the vault alone unless the point of contact is a small one and of not great force; these linear fractures all tend to radiate to the base and into the thinner and weaker parts of the skull. The various types of these fractures of the vault have been outlined in Fig. 1.

It is in these fractures of the vault—whether a linear fracture of the outer table alone or of both tables of the skull, that the underlying brain and intracranial contents frequently escape all damage, either directly or indirectly from hemorrhage and cerebral edema, and thus for these patients the proper designation would be a "fracture of the skull"; if, however, an intracranial lesion has occurred, the presence or not of a fracture of the vault (unless it is a depressed one) is of little or no significance, either in the diagnosis or in the treatment, unless the line of fracture permits the escape of intracranial hemorrhage and excess cerebrospinal fluid into the subcutaneous tissues of the scalp, forming extensive hematomata and thereby facilitating a method of natural decompression which may be sufficient to lower the increased intracranial pressure to such a degree that the cranial operation of decompression and drainage will not be necessary; in these patients, if the overlying scalp is not lacerated or bady bruised and infected, there is little danger of the complication of infection of the underlying hematoma and its extension through the line of fracture intracranially, and yet the greatest care should be used to keep the scalp in an excellent condition of resistance, and then, if the hematoma becomes too tense, it should be aspirated and if necessary, even incised, to facilitate the drainage under the most rigid asepsis.

It should be the routine treatment to shave the entire head of all patients having a severe injury to the vault of the skull; many depressed fractures of the vault are overlooked by not taking this simple precaution. A careful bimanual examination alone may be sufficient; naturally, röntgenograms are of the greatest aid in establishing the presence of many fractures.

In many of the possible depressed fractures of the vault, the overlying scalp is lacerated so that a probe can be gently inserted and a linear fracture of the outer table or of both tables of the skull can be ascertained;

it is, however, a fairly frequent occurrence for the outer table to remain intact while the inner table is fractured and depressed inward; the X-rays are here again of the greatest importance.

In all depressed fractures of the vault, and if the underlying dura is intact, it is the safe procedure, for fear of later trouble, to make a small trephine opening at the edge of the depressed area of bone and, by means of a blunt periosteal or dural elevator, to elevate the depressed fragments, if possible, to their original position; if this attempt is not successful, then the depressed area should be rongeured away. Unless the bone is badly depressed, the dura usually remains intact, and I do not believe it should be opened in these patients unless there are clinical signs of an underlying cerebral lesion—or in the absence of a high intracranial pressure.

In all cases of direct fractures of the vault, if it is at all questionable whether there is a depression of both tables or of the inner table alone of the vault, it is advisable to make a small trephine opening at the edge of the possible depression and thus ascertain its presence or not; if a depression is present, it can be very easily remedied, and if not present, no damage has been done and very little risk has been incurred—other than the usual risk of an anesthetic for several minutes. (A local anesthetic of novocaine suffices in many adult patients.)

The danger of epileptiform attacks occurring after depressed fractures of the vault is much greater than following fractures of the base, undoubtedly due either to small cortical hemorrhages underlying the area of depression and their resulting adhesions of "scar tissue," or to the depression itself rendering the cortex more "irritable" and hence more liable to "neurone explosion." The presence of an increased intracranial pressure in these patients is a definite factor in causing a cortical irritability and thus rendering the patient more susceptible to convulsive seizures.

In patients having not only a depressed fracture of the vault but also marked signs of an increased intracranial pressure with and without a fracture of the base of the skull, then an ipsolateral subtemporal decompression should be first performed, and then through another incision a trephine opening made (as described above) to elevate and even remove the depressed area of the vault; by this method, the general intracranial pressure is safely relieved by the decompression and also the future harmful effects of the depressed area of the vault are avoided. If the depressed area of bone is situated over either the longitudinal sinus or the lateral sinus, then it is frequently wiser not to disturb it, but to rely upon a simple subtemporal decompression to offset any pressure effects of the depressed area of the vault; besides it is most uncommon for epileptiform seizures to occur in the absence of an increased intracranial pressure.

Recent fractures of the vault alone; no signs of an increased intracranial pressure. No operation. Excellent recovery.

A. *Linear fractures of the vault.*

1. Outer table alone.

Case 12.—Linear fracture of the outer table of the vault; laceration of the overlying scalp; multiple injuries. No signs of an increased intracranial pressure. No operation. Excellent recovery.

No. 098.—Michael. Forty-one years. White. Married. Car inspector. Ireland.

Admitted December 31, 1913, Polyclinic Hospital. Referred by Doctor W. S. Bainbridge.

Discharged January 5, 1914—5 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While trying to stop a runaway street car, patient was struck by a plank of wood which knocked the back of his head against the curbing of pavement; unconscious for 20 minutes. Brought to the hospital in the ambulance.

Examination upon admission (50 minutes after injury).—Temperature, 101.6°; pulse, 88; respiration, 20; blood-pressure (not taken). Heavily built man in mild shock. Lacerated wound over left occipital bone; gentle probing reveals a crack and apparently a depression at its lower angle. Contusion of forehead; compound fracture of the nose associated with profuse bleeding and vomiting of blood; Colles fracture of left wrist. No bleeding from the ears; no mastoid ecchymoses. Pupils equal and react normally. Reflexes negative. Fundi negative. Lumbar puncture not performed on account of condition of shock.

Treatment.—Expectant palliative; laceration of scalp shaved widely, cleansed and sutured loosely; 2 drains of rubber tissue inserted.

Examination (14 hours after admission).—Temperature, 101.2°; pulse, 90; respiration, 24; blood-pressure (not taken). Patient conscious and in good condition. Reflexes negative. Fundi negative. Lumbar puncture—clear cerebrospinal fluid under normal pressure (approximately 6 mm.). X-ray (Doctor A. J. Quimby) "shows a definite fracture of the left occipital bone and of its outer table alone; no depression."

Treatment.—Nasal splint applied, also reduction of Colles fracture effected and appropriate splint applied; expectant palliative treatment continued.

Examination at discharge (5 days after admission).—Temperature, 98°; pulse, 80; respiration, 18; blood-pressure (not taken). Laceration of scalp healing nicely—all sutures having been removed to-day. Pupils equal and react normally. Reflexes negative. Fundi negative.

Examination (May 12, 1914—5 months after injury).—No complaints. Reflexes negative. Fundi negative.

Last Examination (July 10, 1918—55 months after injury).—No complaints; patient, however, has become distinctly alcoholic, and this lack of emotional control and instability may be the result of his former head injury—a possible etiological factor. Reflexes negative. Fundi negative.

Remarks.—The condition of this patient and his excellent recovery is a good illustration of the small importance both in the treatment and in the prognosis of patients having cranial injuries with a fracture of the skull—and no signs of an increased intracranial pressure; although this fracture as shown by X-ray was in the most dangerous situation in that it was subtentorially and radiated downward toward the foramen magnum (even though it was only of the outer table alone), yet no definite intracranial signs appeared and particularly those of an increased intracranial pressure producing locally the signs of a direct medullary compression. The presence of a fracture of the vault or of the base in this patient was of no importance in the treatment except to emphasize to the physician the severity of the head injury and the possibility of an intracranial complication and thus the patient would receive more careful attention and examinations than might be otherwise given. Subtentorial fractures are most dangerous fractures of the skull in that intracranial complications and especially direct medullary compression are more liable to occur, and if severe and an early relief of the compression is not effected, then the mortality is very high—undoubtedly higher than in any other form of brain injury.

It is unwise to make prolonged neurological examinations upon these patients when in the initial condition of shock; it is much better judgment to wait until the shock has subsided before attempting thorough and tiring examinations; for this reason the lumbar puncture was not performed upon this patient, even though he was only in mild shock, for fear the condition of shock would at least be prolonged and possibly increased. A blood-pressure record, however, would have been very interesting in this patient as an indication of the severity of the initial shock and its gradual subsidence. The condition of shock is such a big factor in the prognosis of these patients, and particularly in obese adults over middle age, that its estimation should always be recorded and for that purpose naturally, the blood-pressure is a most valuable aid.

In the estimation of shock in these patients upon their admission to the hospital immediately after the injury, the temperature, pulse, respiration and blood-pressure are excellent indices of the extent and severity of the shock then present—this is particularly true as illustrated by the temperature, which is subnormal in the vast majority of these patients, and also of the blood-pressure, which may be lowered to even a hundred and less in the most severe cases, whereas the increased pulse-rate and also respiration-rate are valuable aids in determining the severity of the shock; the general condition of the patient—even an abolition of all reflexes, superficial and deep, should be considered as well as the state of the peripheral circulation—pale, cold, clammy skin, etc. All efforts in treatment should be directed toward the overcoming of the condition of shock, and once the condition of shock has subsided, then the true condition, especially intracranially, can be estimated and treated accordingly; very frequently the signs of increased intracranial pressure (if a large hemorrhage has occurred intracranially before the onset of shock) are overshadowed and submerged by the signs of shock occurring a short time later, and frequently the mild signs of shock are completely obscured by a high intracranial pressure which is possible and which has resulted because the shock was of only mild degree—and therefore the blood-pressure was of sufficient height to permit a large intracranial hemorrhage to occur as the result of a torn vessel. It is most rare, however, for the condition of severe shock to be present and at the same time associated with a high degree of increased intracranial pressure; the one tends to obliterate and make impossible the other in their extreme and severe stages.

It is most important in patients having linear fractures of the vault, either of the outer table alone and particularly if both tables are fractured, that if there is an overlying laceration of the scalp, then this scalp wound should be widely shaved, thoroughly cleansed with green soap after all foreign particles have been extracted as well as possible by means of forceps, especially hair, pieces of stone or dirt, etc., then the tissues swabbed with iodine (great care being taken that no iodine is permitted to enter the line of fracture for fear of producing a severe irritation of the underlying cerebral cortex, especially if the dura has been torn, and thus being the cause of convulsive seizures); two or more drains of rubber tissue should be inserted into the angles of the laceration and then the edges of the wound loosely sutured, so that the tissues will be under no marked tension, and if an infection should occur, the purulent secretion will easily escape through the scalp incision at the points of drainage rather than, being blocked by a too close approximation of the edges of the scalp laceration, the great danger of the infection penetrating into the intracranial cavity through the line of fracture. It is surprising how frequently these "dirty" infected lacerations of the scalp, with or without a fracture of the underlying vault, heal *per primam* with scarcely any redness or edema of the tissues after the wound has been cleansed as described above and its edges approximated loosely and the drains *in situ;* careful shaving of the surrounding scalp is an essential to "first intention" healing of scalp lacerations.

Fractures of the vault of the skull can now be easily located and accurately portrayed by having a series of röntgenograms made at different angles to the vault so that it is possible in this manner to locate small and unsuspected linear fractures—either of the outer table alone or more commonly, of both tables, with or without depression. It is becoming more and more recognized that linear fractures of the vault are of very common occurrence following head injuries of apparently trivial severity; these latent linear fractures of the vault which would not be diagnosed without the aid of the X-ray are of no importance in the treatment, except to impress both the physician and the patient with the necessity of more careful examinations for fear that a definite intracranial lesion, and particularly an increased intracranial pressure, may develop within several days of the injury; that this patient should be confined to bed even though "feeling all right," should be repeatedly examined daily, especially neurologically and ophthalmoscopically, and that the patient should not re-enter the former active, vigorous life until a period of at least several weeks has elapsed. The vast majority of these patients having simple linear fractures of the vault do not develop marked signs of increased intracranial pressure and therefore they make excellent recoveries with the expectant palliative treatment alone. It must always be remembered that the fracture of the skull, and particularly the simple linear fractures of the vault and also of the base, are possibly of the least importance and the smallest factor in the treatment and prognosis of cranial injuries—the most important factor being the presence or not of high intracranial pressure, whether due to intracranial hemorrhage or cerebral edema.

2. Both tables of the vault.

CASE 13.—Linear fracture of both tables of the vault; no signs of an increased intracranial pressure. No operation. Excellent recovery.

No. 931.—John J. Sixty-one years. White. Married. Boat-builder. United States.

Admitted November 22, 1917, Polyclinic Hospital. Referred by Doctor Alexander Lyle.

Discharged December 16, 1917—24 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While crossing the street, patient was struck by an automobile; unconscious. Brought to the hospital in the automobile.

Examination upon admission (15 minutes after injury).—Temperature, 98°; pulse, 86; respiration, 28; blood-pressure, 140. Unconscious and in mild degree of shock. Hematoma over left squamo-parietal suture. No bleeding from nose, mouth or ears. Pupils equal and react normally. Reflexes obtained with difficulty, but equal; no Babinski; abdominal reflexes could not be elicited. Fundi negative. Lumbar puncture—cerebrospinal fluid clear and under normal pressure (9 mm.).

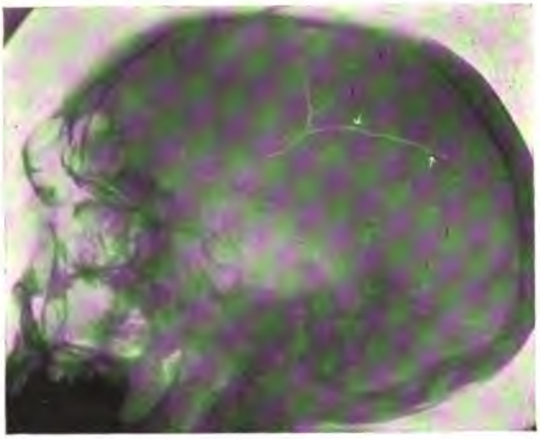


FIG. 54.—Transverse linear fracture of left parietal bone with no signs of an increased intracranial pressure; naturally no operation. Excellent recovery.

Treatment.—Expectant palliative.

Examination (14 hours after admission).—Temperature, 99.6°; pulse, 80; respiration, 22; blood-pressure, 144. Conscious but confused mentally. Extensive hematoma of left side of the head with an underlying definite line of tenderness; hematoma apparently enlarging. Pupils negative. Reflexes active but otherwise negative. Fundi negative. X-ray (Doctor G. W. Welton) "shows a linear transverse fracture of left parietal bone; also an apparent dislocation of the last 3 coccygeal bones" (Fig. 54).

Treatment.—Expectant palliative; scalp over hematoma carefully shaved and cleansed for fear of infection (apparently an extradural hemorrhage was being drained through the underlying fracture of the bone).

Examination (6 days after admission).—Temperature, 99.2°; pulse, 74; respiration, 20; blood-pressure, 144. Markedly improved; entirely conscious and well oriented. Hematoma over left parietal region decreasing in size and becoming less tense. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi negative.

Examination at discharge (24 days after admission).—Temperature, 98.8°; pulse, 76; respiration, 20; blood-pressure, 142. Feels well except for pain in the coccyx, which has considerably lessened within the past 3 days. Hematoma has entirely disappeared; a definite line of tenderness, however, persists at the site of the fracture, which can be accurately palpated. Reflexes active but otherwise negative. Fundi negative.

Examination (May 10, 1918—5 months after injury).—In excellent health and no complaints; feels possibly better now than before the injury—due to the fact that he is taking excellent care of himself, having had his first vacation in 14 years. Reflexes active but otherwise negative. Fundi negative.

Last Examination (January 20, 1919—14 months after injury).—No complaints, and is working as hard as formerly. Reflexes active but otherwise negative. Fundi negative.

Remarks.—The presence of a hematoma over the lower left parietal area with a fracture of the underlying bone as revealed by the röntgenogram, and the gradual enlargement of this hematoma until it became very tense, would point to the hematoma as being the result of blood extruding through the fracture of the bone and thus preventing an increase of the intracranial pressure by its escape—a sort of natural decompression; the absence of blood in the cerebrospinal fluid at lumbar puncture would tend to indicate that the bleeding was extradural entirely, and yet this cannot be stated with certainty because it is frequently ascertained at operation that a subdural and subarachnoid hemorrhage in fluid form can be present in fairly large amount and yet the cerebrospinal fluid at lumbar puncture is clear—both macroscopically and microscopically—up to the time of the cranial operation. This observation, however, has only been confirmed in patients having a marked increase of the intracranial pressure (as naturally these are the only patients operated upon), and whether in these patients having subdural bleeding and yet no blood in the spinal cerebrospinal fluid there is a blockage of the intracranial spinal fluid at the foramen magnum so that in some patients the intracranial blood can therefore not descend into the spinal canal—this explanation cannot be stated with certainty; in patients, however, who have no marked increase of the intracranial pressure and also no blood in the spinal cerebrospinal fluid, whether there is free blood or not in the intracranial cerebrospinal fluid cannot be demonstrated, but if the blockage is due to an increased pressure at the foramen, this factor would not be present in these patients.

It would seem that here again we have a sort of "natural" decompression resulting from the intracranial blood being able to escape through the line of fracture of the vault and thus forming a hematoma within the overlying scalp; as no blood was demonstrated to be present in the cerebrospinal fluid at lumbar puncture, it would seem that either the amount of intracranial hemorrhage had been small and quickly absorbed, or that so much intracranial free blood had escaped through the fracture and had been absorbed at the site of the hematoma so that in this manner the sig of a definite increase of an intracranial pressure had been avoided. In cases similar to this, the fracture of the vault is to be hoped for, and instead of it increasing the severity of the condition and making the prognosis more grave, it on the contrary increases the patient's chances of recovery without an operation—not only as to an immediate recovery but as to the patient's ultimate good health. It is in these patients, however, that if there is any question of an infection of the hematoma overlying the fracture of the vault, then there should be no hesitancy in at least aspirating the hematoma through a "clean" area of the scalp repeatedly, and if necessary a small incision made and a drain of rubber tissue inserted—for fear that the hematoma might otherwise become infected and its extension through the line of fracture intracranially would result in a fatal meningitis or a later brain abscess. Any contusion or abrasion of the overlying and adjacent scalp should be most carefully cleansed and a wet bichloride (1–5000) dressing or a gauze dressing with a mild solution of alcohol applied.

3. Linear fracture of both tables of vault of skull associated with high intracranial pressure due to large extradural hemorrhage. Incision of scalp and drainage. Recovery.

CASE 14.—Acute severe brain injury associated with a wide linear fracture of the skull and with signs of high intracranial pressure due to a large extradural hemorrhage; left hemiplegia. Scalp incision and the partial removal of the extradural hemorrhagic clot through the fracture of the vault. Recovery.

No. 009.—Yuan. Thirty-five years. Yellow. Married. Eldest son of the first President of China. China.

First Examination (May 12, 1912—10 weeks after injury).—Summer home of Emperor of China at Yangteh Fu, Honan, China. Referred by Captain Tsai Tin Kan.

Operation (May 22, 1912—11 weeks after injury).—Removal of extradural clot through overlying linear fracture of vault.

Last examination June 6, 1912, 14 days after operation.

Family history negative.

Personal History.—Negative; always well and strong.

Present Illness.—Ten weeks ago while riding horseback, patient was thrown to the ground, striking the back of his head against a large boulder; immediate loss of consciousness; no bleeding from the nose, mouth or ears; diffuse boggy hematoma over entire top of head (no signs of it at examination now except an indistinct bluish area over the right parietal bone). Patient regained consciousness upon the sixth day after the injury and it was then learned that there was a complete left hemiplegia. Patient complained of severe frontal and occipital headache and dizziness.

Treatment has been of the expectant palliative type and during the past 10 weeks the general condition of the patient has steadily improved with the exception of the continued complete left hemiplegia.

Examination (10 weeks after injury).—Temperature, 98.8°; pulse, 66; respiration, 18; blood-pressure, 136. Fair development and nourishment. Conscious; complains of dull frontal and occipital headache. No aphasia nor phasia (patient being right-handed, as were his parents and grandparents). Complete left hemiplegia—left side of face being possibly less paralyzed than the left arm and left leg, which could not be moved—not even a finger or toe of the left hand or left foot. No marked impairment of sensation of the left side of the body, except a general dulness of the sensation to light touch and pin-pricks; these sensations were all delayed, which was considered at the time to be due to a lack of concentration and a slight mental retardation of the patient. Owing to difficulty of language, no accurate estimation of patient's mental and emotional status could be ascertained, although he apparently appeared to be normal—and the Chinese doctors considered his mentality "ding how" (excellent). Some impairment of the stereognostic sense of the left hand—not present in the right hand. No apraxia. Definite tenderness over the right parietal area; no depression palpable. Pupils—right larger than left and reacts to light sluggishly. Reflexes: patellar—left very much exaggerated, patellar clonus being elicited; left inexhaustible ankle clonus and left Babinski but only right exhaustible ankle clonus and a suggestive right Babinski; left abdominal reflex absent, right inactive; deep reflexes of left arm and left masseteric reflexes markedly increased. Fundi—retinal veins full, tortuous and almost buried in edematous retinæ about the optic disks; nasal halves and temporal margins blurred by edema but no measurable swelling ascertained—that is, a papilledema but not to the extent of producing "choked disks." Lumbar puncture—clear cerebrospinal fluid under high pressure (approximately 20 mm.). No X-ray picture could be taken—there being no X-ray machine nearer than Pekin, a distance of 400 miles.

Treatment.—In the presence of the general signs of such high intracranial pressure and confirmed by the more accurate ophthalmoscopic and lumbar puncture tests, and in the presence of the localizing signs of a left hemiplegia, slight sensory impairment and indefinite astereognosis, the diagnosis was naturally one of extensive intracranial hemorrhage compressing the right cerebral hemisphere, and most probably an extradural one, especially in the absence of blood in the cerebrospinal fluid at lumbar puncture and of the signs of cortical irritation such as convulsive seizures. The fact that these pressure and localizing signs had persisted for a period of ten weeks following the injury in spite of the expectant palliative method of treatment, it was considered most improbable that this intracranial hemorrhage could be absorbed by natural means; it was, therefore, advised that the patient should be transported to Pekin, where a cranial operation could be performed in a hospital under the modern conditions of asepsis and assistance. The family, and particularly the mother of the patient, would not consider the journey to Pekin so that, after a period of 10 days, consent was finally obtained for an incision of the scalp overlying the right parietal area, using cocaine anesthesia; it was thought a small trephine opening could be made in this area and, if an extradural hemorrhage was present, it could be removed, or at least a large part of it, through this small opening—a comparatively simple procedure and devoid of danger to the patient. After much discussion, delay and differences of opinion (there being nine doctors in consultation), a permission for this operation was finally obtained.

Operation (11 weeks after injury).—Scalp incision with removal of extradural clot through the line of fracture of the overlying vault (cocaine anesthesia alone being used); patient's head had been carefully shaved and cleansed with soap and alcohol; under cocaine anesthesia a small vertical incision of two inches was made over the right parietal area; upon retraction, much "free" blood clot was found in the subcutaneous tissues and protruding through the pericranium beneath the fronto-occipital aponeurosis, giving the latter a bluish tint; upon incising it, there was exposed a fracture of the underlying parietal bone, over one-eighth of an inch in width and extending antero-posteriorly about one and a half inches from the longitudinal sinus which it paralleled. Protruding through this wide line of fracture was a bluish blood-clot of the consistency of soft gelatine, and upon removing the outer portion with small forceps the underlying portion of blood-clot was forced upward by the increased intracranial pressure and thus it was exuded extracranially; as this clot was removed by the forceps from the line of fracture, more blood-clot was forced into view and in this simple manner almost 4 tablespoonsful of semi-solid blood-clot were removed; then dark blood of the consistency of currant jelly oozed through the line of fracture and over 2 ounces welled out at this time. A small probe now inserted through the line of fracture revealed the underlying dura depressed about 1 cm. beneath the inner table of the vault of the skull, but apparently it was intact. Two small strips of rubber cut from a glove and thoroughly boiled were now inserted as drains just through the line of fracture, and came out at either end of the scalp incision, which was closed loosely by 5 silk sutures. Wet gauze dressings (thoroughly boiled) were now applied and a large head bandage. Duration—40 minutes.

Post-operative Notes.—The wound continued to drain a large amount of dark syrupy blood which finally ceased on the third day after operation; the rubber drains were now removed. Patient had almost immediate relief of the severe headache and on the fourth day after operation, he found it possible to move the toes of the left foot and the fingers of the left hand. The improvement daily progressed so that on the eighth day after operation he was able to move both the left arm and the left leg, and on the twelfth day after operation he was able to move about with improvised crutches; normal sensation over the left side of the body and there was no astereognosis to be elicited in the left hand. Operative incision healed *per primam.*

Last Examination (14 days after operation).—Temperature, 98.6°; pulse, 74; respiration, 20; blood-pressure, 134. Perfectly conscious; apparently no impairment of mentality nor emotional instability. No complaints except a feeling of soreness at the site of the scalp incision, which has healed perfectly. Only slight weakness of the left arm and left leg can be elicited by special tests, and no weakness of the left side of the face; no sensory impairment; no astereognosis. Pupils equal and react normally. Reflexes: patellar—exaggerated, left more than right; only left exhaustible ankle clonus and a tendency to left Babinski, but normal right reflexes; abdominal reflexes depressed, left more than right; deep reflexes of left arm greater than right; masseteric reflexes—both sides equal. Fundi—retinal veins enlarged, right possibly more than left; nasal margins only of both optic disks blurred by edema, right possibly more than left. Patient is able to walk a number of steps without crutches and is regaining very rapidly the strength and use of left arm and left leg.

Treatment.—Daily massage and exercises of left side of body; general hygienic measures; triple bromides, grains x, three times a day, after meals, to lessen the cortical irritability and thus decrease the danger of future convulsive seizures.

Report (February 28, 1913—9 months after operation).—Letter from Mr. J. C. Wang, secretary to the President: "Your patient has made an excellent recovery; there is still some weakness of the left leg but his left arm is now well. He is leading a quiet life in their home in Honan; he is planning to come to Pekin this summer."

Report (September 16, 1914—27 months after operation).—Patient had a convulsive seizure, 25 months after the operation, and apparently beginning in the left leg; no loss of consciousness in the first three Jacksonian convulsions, but he has now had six general convulsive seizures with loss of consciousness. "Will an operation upon his head help him now get over this disease?"

Last Report (49 months after operation).—On the same day that his father was found dead under rather suspicious circumstances, this patient—the President's eldest son—was also found dead in bed, presumably from poison.

Remarks.—This patient was a most instructive one from many points of view: for an increased intracranial pressure of such high degree to persist for a period of eleven weeks following the cranial injury and yet no marked mental and emotional impairment to result as well as no signs of a definite medullary compression and even loss of medullary compensation to the degree of a medullary edema, is most unusual; these patients, having an increased intracranial pressure of such marked degree, unless this pressure is relieved comparatively early, gradually become exhausted so that a sudden onset of medullary edema frequently occurs; the fact that this patient was not beyond middle age was of great value to him.

This is the only patient in this series of brain injuries in whom it was possible to relieve the intracranial hemorrhage by means of removing the extradural blood-clot through a linear fracture of the vault; there are many patients who "decompress" themselves by the intracranial "free" blood escaping through the line of fracture extending through either ear, the nares or through a linear fracture of the vault, producing a hematoma of varying degree; but it is most rare, I believe, and I cannot find a similar case in the literature of brain injuries, where it was possible to remove clotted and semi-solid blood through a single line of fracture of the vault—clotted blood of the amount sufficient to relieve the intracranial pressure and direct compression so that the resulting condition of paralysis and other definite cerebral impairment was markedly benefited. The reason for this is due chiefly to the fact that it is a most rare occurrence for a linear fracture of the vault to be so widely dilated that it is possible for clotted blood to be extruded by the resulting increased intracranial pressure, and unless the fracture of the vault is a comminuted one, then only the blood in fluid form can escape to form the hematoma and the clotted blood is unable to escape owing to the linear fracture being usually but a narrow line—not wider than a mere crack into which it would be most difficult to pass a sheet of the thinnest paper. In this patient, however, the linear fracture undoubtedly resulted from the "bursting" effect of the cranial injury, and, as Doctor O. H. Schultze has frequently demonstrated in this type of fracture of the vault, the central portion is more widely dilated than either end, and in this particular patient the fracture was separated widely, similar to the diastasis of suture lines following cranial injuries. The bogginess over the top of the head following the injury in this patient was undoubtedly due to the escape of fluid blood through this line of fracture; the extradural hemorrhage being of slow but continuous formation, soon clotted so that within 12 to 24 hours following the injury no more extradural blood could escape through the line of fracture and thus there was formed this large extradural clot lying directly beneath the site of the fracture of the vault—possibly the size of a flattened orange, and directly overlying the motor area of the right cerebral hemisphere. It is very probable, therefore, that the left hemiplegia did not occur until possibly 24 hours after the cranial injury when the clotting of the extradural hemorrhage prevented its being extruded through the line of fracture and thus the intracranial pressure was gradually increased as the dura was slowly dissected, as it were, from the inner table of the vault at the site of the fracture until the extradural hemorrhage had reached its large size and the bleeding vessel or vessels had become thrombosed; as the result of this increasing intracranial pressure, the patient did not regain consciousness as early as is usual for these patients and when he did, then it was noticed for the first time that the left side of the body was paralyzed.

This patient should naturally have had a right subtemporal decompression first performed to relieve the increased intracranial pressure, and then a removal of an area of bone, preferably a small osteoplastic flap over the right parietal area—the site of the extradural hemorrhage. In this manner, the increased intracranial pressure could have been entirely removed and the extradural clot completely evacuated so that the danger of future complications would have been very slight indeed. It being impossible, however, to transport this patient to a hospital where such an operation could be safely and successfully performed, and it not being possible to obtain the consent of the patient's mother for a more extensive operation than a mere exploratory incision of the scalp overlying the site of the cerebral lesion, it was therefore obligatory, if anything was to be attempted to improve the condition of this patient, that it should be of the most incomplete and superficial character—but as safe as the conditions would warrant. The instruments and operative gauze sponges and dressings were all boiled for a period of 15 minutes and used wet, and the operator was the only one to touch the instruments and the gauze sponges and dressings with his sterile gloved hands; the two retractors with long handles were held by two Chinese doctors who were not surgically "clean" but known to be "dirty" and who were therefore not permitted to touch anything; in this manner, the danger of infection of the wound was minimized, although the general conditions in the room, the patient's bedroom, were bad in that the mother insisted that a number of the household be present during the operation, "to make sure that everything would be all right."

The onset of the convulsive seizures 25 months following the operation merely indicates that the patient only apparently recovered and that there was undoubtedly not only a persistent increased intracranial pressure due to the resulting cerebral edema, but that the extradural hemorrhage had only been partially removed, so that it produced an irritation of the underlying cortex and eventually the convulsive seizures. It is most unfortunate that the more extensive operation of right subtemporal decompression and then the removal of the extradural clot could not have been performed in a hospital under modern aseptic conditions, or an attempt made even in the patient's home to perform this operation with proper assistance and under strict aseptic precautions; it was impossible, however, to obtain the consent of the mother for this more extensive procedure (even though the patient himself desired it, but, as he said, "I must obey my mother"—a remarkable instance of the filial obedience of the Chinese as a race), and it required over a week to obtain the permission of the mother even for this minor exploratory incision of the scalp; at the time, it was still hoped that, after the scalp incision had been made, a small trephine opening could then be performed and the condition, if an extradural hemorrhage, could be thus drained with little or no risk to the patient; the presence of such a wide fracture of the underlying vault was not even suspected. Since this operation, it has been demonstrated upon a number of patients that a trephine opening of the vault can be made under local anesthesia of the overlying scalp with very little pain to the patient until the dura is reached; the dura itself is very sensitive. (Upon my return to Pekin following the operation, I gave my word to the patient's father, the President of China, and at his request, that I should not mention anything to anyone about the operation upon his son—his eldest boy and the probable successor to the throne. The death of both the father and on the same day of the patient himself, frees me of this promise so that I am now publishing in book form a brief account of my experience in the summer home of this Chinese potentate amid Oriental splendor.)

B. *Recent depressed fractures of the vault.*

1. No operation.

Case 15.—Recent depressed fracture of the vault. Operation refused. Recovery doubtful.

No. 13.—Gus. Fifty-four years. White. Married. Longshoreman. Norway.

Admitted February 5, 1914, Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Discharged February 7, 1914—2 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While loading a ship, patient was struck by the chain of a crane; unconscious for several minutes. Brought to the hospital in the ambulance.

Examination upon admission (1 hour after injury).—Temperature, 100.4°; pulse, 100; respiration, 20; blood-pressure, 126. Comatose; not alcoholic. Two stellate lacerations over right side of head; careful probing of upper wound reveals 2 small depressions of 1 cm. in diameter—outer table of the skull only considered to be involved. No bleeding from nose, mouth or ears; no mastoid ecchymosis. Pupils equal and react normally. Reflexes negative. Fundi—retinal veins possibly enlarged; nasal margins of optic disks slightly blurred by edema. Lumbar puncture not performed. X-ray (Doctor A. J. Quimby)—"depressed fracture (punctate) of right parietal bone." Urine negative.

Treatment.—Expectant palliative; scalp laceration widely shaved, cleansed and loosely sutured; 2 drains of rubber tissue inserted.

Examination at discharge (2 days after admission).—Temperature, 98.6°; pulse, 80; respiration, 20; blood-pressure, 138. Patient has refused all treatment and insists upon "going home"; refuses to permit the elevation of the depressed area of bone to prevent future complications. Reflexes negative. Fundi—retinal veins still enlarged; edematous blurring along the nasal margins of both optic disks.

Treatment.—Patient advised to refrain from work for at least a month and to return at frequent intervals for examination.

Examination (May 28, 1914—3 months after injury).—No complaints; working as usual. Reflexes negative. Fundi negative. Small depressed area of bone over right parietal bone can be easily palpated; not tender.

Examination (September 2, 1916—31 months after injury).—No complaints; "as well as ever." Reflexes negative. Fundi negative.

Examination (June 17, 1918—52 months after injury).—No complaints; patient, however, is decidedly alcoholic. Reflexes—knee-jerks obtained with difficulty, requiring reinforcement; no Babinski; abdominal reflexes absent. Fundi—general suffusion of entire retinæ; no definite edema about optic disk margins.

Remarks.—Whether this patient will remain "perfectly well" is very doubtful, particularly if complicated by the factor of alcoholism. A definite depressed fracture of the vault is undoubtedly a source of local irritation to the underlying cerebral cortex, but if the patient is of the stable emotional type and the irritation is not one of severe degree, then it is possible for no definite signs of cortical irritability to result, especially if he refrains from dissipation of all kinds, and particularly alcoholic indulgence. But in a patient at all unstable, or if the cortical irritation resulting from the depressed area of bone is marked, and especially if alcoholism becomes pronounced, then the danger of convulsive seizures and of the milder forms clinically of cortical irritability is very great indeed. As this patient has no definite signs of an increased intracranial pressure (as revealed by the ophthalmoscope), these fundal changes are probably due to a slight chronic cerebral edema resulting from the cranial injury in the presence of alcoholism, so that the risk of emotional disturbances at least and of epileptiform seizures, especially after a prolonged continuance of the daily use of alcohol, makes the prognosis very doubtful, and unless the habits of the patient change markedly, the danger of cerebral complications is a very great one.

It is hoped that this patient can be followed during the next few years and the condition reported later.

2. Removal of depression alone.

Case 16.—Depressed fracture of vault; no signs of increased intracranial pressure. Operative removal of depression only. Excellent recovery.

No. 510.—Edward. Forty-five years. White. Married. Laborer. Ireland.

Admitted February 8, 1916, Polyclinic Hospital.

Operation February 9, 1916. Removal of depressed bone.

Discharged February 21, 1916—12 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While digging a ditch, patient was struck over the left side of the head by a shovel; no loss of consciousness. Brought to the hospital in the ambulance.

Examination upon admission (30 minutes after injury).—Temperature, 98.6°; pulse, 74; respiration, 22; blood-pressure, 144. Well-developed Irishman in good condition; perfectly conscious; jokes regarding his condition as an amusing incident; no signs of shock. A curved laceration of the scalp of almost two inches, extending from the left parietal crest downward toward the left ear; exploration of wound with probe and gloved finger reveals a very definite depression of the underlying bone. No signs of cortical irritation or of paralysis; no sensory impairment. No bleeding from nose, mouth or ears; no mastoid ecchymoses. Pupils equal and react normally. Reflexes negative. Fundi negative. X-ray (Doctor W. H. Stewart)—"definite depression of left parietal bone near the squamo-parietal suture."

Treatment.—Head prepared for operation; entire scalp closely shaved, laceration cleansed with green soap, and a wet bichloride (1–5000) dressing applied to laceration, and a green soap poultice to the entire scalp, including the ears.

Operation (14 hours after admission).—Removal of depressed area of bone: under ½ per cent. novocaine local anesthesia, each angle of the original wound was extended about one inch, the scalp retracted and the underlying bone bared of periosteum; a small button of bone, 1 cm. in diameter and similar to a trephine button, was exposed; it was completely broken off from the surrounding vault and was depressed against the dura to a depth of 1 cm. Sufficient bone was rongeured away to permit the depressed fragment to be removed; dura not opened; underlying cortex apparently negative. Two drains of rubber tissue inserted down to dura and incision closed loosely with black silk; usual gauze dressing and head bandage applied. Duration—18 minutes.

Post-operative Notes.—Uneventful operative recovery; healing *per primam.*

Examination at discharge (13 days after admission).—Temperature, 98.8°; pulse, 80; respiration, 22; blood-pressure, 138. Condition excellent. Laceration and operative incision healed perfectly. Reflexes negative.

Fundi negative. A second X-ray picture "reveals a circular bony defect in the posterior left parietal area" (Fig. 55).

Examination (June 20, 1917—16 months after injury).—No complaints; working as before the injury; has been worried by pulsation at site of operation, and this is now explained to him as being perfectly normal. Reflexes negative. Fundi negative.

Last Examination (July 12, 1918).—No complaints. Site of removal of depressed area of bone depressed and very firm, as though being filled in with new bone tissue. Reflexes negative. Fundi negative.

Remarks.—It is interesting to note the ease with which this operation was performed under local anesthesia and with what little pain the bone could be rongeured away; pulling the dura caused a definite painful sensation over the entire left side of the head and face—left trifacial nerve irritation. The use of local anesthesia in this case suggests the possibility of a still wider application of it for selected cases of cranial conditions in the future. This patient had developed no signs or symptoms of his head injury up to the time of operation and yet it was considered most advisable to remove the depressed area of bone for fear of future complications—particularly headache and the signs of cortical irritation, etc.

It is very seldom that depressed fractures in adults can be elevated—unless both tables of the vault are entirely fractured at the periphery of the depression and in these patients it is not wise to leave these "islands" of bone—they should always be removed for fear of future complications such as necrosis, the danger of infection and of a definite displacement of the fragments themselves. In the vast majority of patients the depressed area of the vault must be removed and in most cases it is necessary to make a small trephine opening at the periphery of the depressed area in order to enlarge the bony opening so that the depressed fragments can be removed: naturally, if the underlying dura is torn it should be, if possible, sutured; it may be necessary to perform a subtemporal decompression first in order to lessen the increased pressure so that this torn dura beneath the depressed fracture of the vault can be easily and safely repaired.

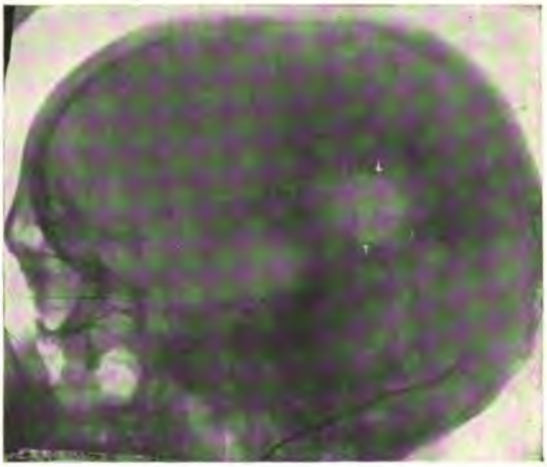


FIG. 55.—Circular bony defect of removal of depressed area of posterior portion of left parietal bone. No increase of intracranial pressure. Excellent recovery.

In these acute depressed fractures of the vault which have a laceration of the overlying scalp, it is of the greatest importance that this laceration should be cleansed most thoroughly with green soap and then the tissues "touched" with iodine and thus the danger of infection minimised as much as possible; if there is still almost certain evidence that these lacerated tissues are infected or will become infected, then it is better surgical judgment to excise them until all the infective material and possibly infected tissues are removed. The danger of a possible infection extending down through the fracture of the vault and, if the dura is torn, the great risk of a purulent meningitis and also cerebral abscess developing, should always be remembered; many of these patients develop an osteomyelitis of the underlying bone and if the dura is not torn, it frequently acts as a barrier to the direct extension of the infection intracranially, although there is always the risk of meningitis and less frequently of abscess formation developing at a later date.

A general anesthetic was used for most of these patients having simple depressed fractures of the vault, but I now believe that local anesthesia (preferably a weak novocaine solution) would be advisable. Naturally, if it was necessary to perform a more extensive operation than planned originally, then general anesthesia could be administered. Many of these patients are alcoholic and in poor condition physically, so that a general anesthetic should be avoided as much as possible for fear of pulmonary complications, etc.

There are certain small depressed fractures of the vault occurring in the occipital region, and particularly about the sigmoid and lateral sinuses, and also over the torcula, when it is better surgical judgment to "let them alone" rather than to attempt their removal and possibly have the complication of severe hemorrhage; naturally, if there are signs of their presence intracranially, then they should be removed and the injured sinus packed or ligated. It is surprising that the longitudinal sinus is rarely torn by depressed fractures of the vault overlying the sinus, and that these areas of depressed bone can usually be removed without any complication of hemorrhage from the sinus itself. I believe that all depressed fractures of the vault should be elevated or removed, whenever possible surgically, for fear of future complications.

Recent Mild Brain Injuries Associated with a Fracture of the Base of the Skull and with Signs of an Increased Intracranial Pressure. No Operation.

In many patients having severe cranial injuries, the fracture of the base of the skull is the main pathology resulting, and therefore these cases may be labelled as fractures of the base of the skull. The intracranial contents may be little if any damaged, so that if an increased intracranial pressure does occur it is usually of such a mild degree that the expectant palliative treatment alone is sufficient to obtain an excellent recovery, both of life and of future normality; if, however, the intracranial lesion has produced a marked increase of the intracranial pressure, whether due to hemorrhage or cerebral edema, then the greatest care must be exercised in determining whether the expectant palliative treatment will be sufficient or the necessity of the mechanical relief of this increased pressure by means of the cranial operation of decompression in order to secure the best result. The presence of the fracture of the base is really of little concern in the treatment unless the discharge of blood and cerebrospinal fluid through the line of fracture into the nose or ears materially lessens the increased intracranial pressure; with appropriate precautions in these latter patients, the danger of infection is slight.

The diagnosis of cranial injuries sufficient to produce a fracture of the base of the skull is frequently facilitated by the line of fracture passing into either middle ear with a rupture of the tympanic membrane, or into the cribriform plate of the frontal bone so that blood, and especially cerebrospinal fluid, are discharged into the external auditory canal and nares respectively; fractures of the base subtentorially extending into the naso-pharynx, such as those of the basilar process of the occipital bone, are also most serious ones on account of the great danger of infection, while fractures into or around the foramen magnum may cause so much hemorrhage subtentorially that a direct compression of the medulla itself frequently results. Fractures of the base extending into either orbit usually cause an escape of blood into the tissues of the orbit and thus ecchymosis of extreme degree may occur; subconjunctival hemorrhages are also of common occurrence but they are not pathognomonic of an adjacent fracture of the orbital bones. Röntgenograms are rarely of value in the diagnosis of basal fractures; if, however, positive pictures can be obtained—particularly of the occipital bone about the foramen magnum by means of the open mouth, the gravity of the patient's condition could thus be early recognized and all appropriate measures instituted to lessen the danger of subtentorial complications.

The majority of the patients having a fracture of the base of the skull do not disclose the signs of a marked increase of the intracranial pressure and therefore they make excellent recoveries under the expectant palliative treatment alone. It is in those patients, however, in whom the ophthalmoscope reveals an early papilledema with blurring of the nasal halves and even the temporal halves of the optic disks, and the spinal mercurial manometer registers the pressure of the cerebrospinal fluid as being 16 mm. and even higher—these are the patients for whom the expectant palliative treatment is not a safe and rational method of treatment—not only as concerns the immediate life of the patient but also his future condition of good health; in these patients, the operation of decompression and drainage is the safer procedure to obtain the best immediate and ultimate result—whether the increased intracranial pressure is due to hemorrhage or cerebral edema. Naturally, cerebral contusions and lacerations, either directly overlying the line of fracture or more usually of the "*contre-coup*" type to the point of contact—these complications and their manifestations of paralysis, impaired sensation, etc., can be improved by the treatment but not wholly eradicated on account of the non-regeneration of cerebral tissue—the cortical nerve cells and the non-medullated subcortical nerve fibres; the nerve tissues not primarily destroyed but merely functionally compressed—these tissues can regain their normal function with the appropriate treatment so that a marked improvement can thus be obtained.

Recent fractures of base of the skull; signs of increased intracranial pressure. No operation. Recovery.

A. *Mild increase of the intracranial pressure.*

a. Excellent recovery.

CASE 17.—Fracture of base of skull; signs of a mild increase of the intracranial pressure. No operation; repeated lumbar punctures. Excellent recovery.

No. 344.—Thomas. Thirty-one years. White. Single. Clerk. U. S. Admitted August 23, 1915, Polyclinic Hospital. Referred by Doctor John A. Bodine.

Discharged September 12, 1915—19 days after injury.

Family history negative.

Personal history negative.

Present Illness.—After coming out of a store, patient was struck over the head by an unknown man with a baseball bat; knocked down (bat breaking) and was unconscious for several minutes; brought to the hospital in the ambulance.

Examination upon admission (25 minutes after injury).—Temperature, 98.2°; pulse, 80; respiration, 24; blood-pressure, 132. Semiconscious and in very little shock. Laceration of scalp over left frontal bone and also over the occipital protuberance. Profuse hemorrhage from nose and bloody cerebrospinal fluid dropping from right ear; right mastoid ecchymosis. Pupils equal and react normally; nystagmoid twitches to right. Reflexes negative. Fundi negative. Lumbar puncture—cerebrospinal fluid bloody and under a slight pressure (approximately 10 mm.).

Treatment.—Expectant palliative; lacerations of scalp shaved widely, thoroughly cleansed and loosely sutured with fine black silk; 2 drains of rubber tissue inserted.

Examination (30 hours after admission).—Temperature, 100.2°; pulse, 84; respiration, 24; blood-pressure, 136. Conscious; complains of severe headache "all over." Bleeding from nose has ceased, but cerebrospinal fluid appears at intervals in the right external auditory canal. Pupils equal and react normally; nystagmoid twitches to right have disappeared. Reflexes negative. Fundi—retinal veins enlarged; an indistinct blurring of the nasal margins of both optic disks. Lumbar puncture—cerebrospinal fluid bloody and under slightly increased pressure (approximately 10 mm.). X-ray (Doctor J. A. Quimby)—"no fracture of the skull can be ascertained."

Treatment.—Expectant palliative; lumbar puncture with drainage of 15 c.c. of cerebrospinal fluid was performed each day for 3 successive days—the last day, the cerebrospinal fluid was straw-colored and under a normal pressure (9 mm.); the headache was each time temporarily improved.

Treatment.—Expectant palliative continued.

Examination at discharge (19 days after admission).—Temperature, 98.6°; pulse, 78; respiration, 18; blood-pressure, 138. General soreness about head but otherwise no complaints; occasional headache. Laceratio of scalp healed perfectly. Otoscopic examination reveals a tear of th posterior quadrant of right tympanic membrane; no cerebrosp discharging and no middle ear infection occurred. Hearing—right ear impaired; bone conduction greater than air conduction. Reflexes negative. Fundi practically negative within physiological limits.

Examination (January 10, 1917—16 months after injury).—No complaints. Reflexes negative. Fundi negative. Hearing less acute in right ear; bone conduction equals air conduction.

Last Examination (July 12, 1918—35 months after injury).—Patient thinks he is as well as ever. Reflexes negative. Fundi negative. Hearing possibly less acute in right ear, although bone conduction is less than air conduction.

Remarks.—On account of the profuse and continuous discharge of cerebrospinal fluid from the right ear, it was thought advisable to perform repeated lumbar punctures and drainage of the excess cerebrospinal fluid and thus tend to lessen the discharge of cerebrospinal fluid through the ear and thereby decrease the chances of infection through the ear. Any *prolonged* discharge of cerebrospinal fluid through the ear is a very dangerous means of lessening the increased intracranial pressure on account of the danger of infection. It was interesting to observe the cessation of the discharge of cerebrospinal fluid from the ear after each lumbar puncture.

Although this patient was in the condition of mild shock with a temperature of 98.2°, the pulse of only 80 and a blood-pressure of 132 would indicate that there was an increased intracranial pressure and that the signs of mild shock, beside the subnormal temperature, should have included an increased pulse-rate of 100 and above and a lowered blood-pressure to 110 and below, if it were not that these mild signs of shock had been overshadowed and submerged by a definite increase of the intracranial pressure; this was disclosed accurately by a later ophthalmoscopic examination (which rarely reveals signs of an increased intracranial pressure within 6 hours after the injury and chiefly due to this initial period of shock) and by the measurement of the pressure of the cerebrospinal fluid at lumbar puncture.

In this patient the bloody cerebrospinal fluid as withdrawn at lumbar puncture was associated with the discharge of cerebrospinal fluid from the ear; this association, however, does not necessarily follow because it is of frequent occurrence to have an intracranial subdural and subarachnoid "free" hemorrhage with or without the discharge of cerebrospinal fluid from the ear and yet the cerebrospinal fluid at lumbar puncture is clear and remains clear.

The definite improvement following each therapeutic lumbar puncture with removal of 15 c.c. of cerebrospinal fluid is very impressive, not only from the subjective benefit but from the fact that at least some of the "free" hemorrhage must have been drained away that otherwise could not have been absorbed by natural means, and therefore a larger residue of connective-tissue formation and adhesions would have been possible and thus the danger of future complications increased.

The gradual improvement of the hearing of the right ear of this patient is so frequently the history of many of these traumatic cases; immediately following the injury with the impairment of hearing being referred to the mid-
account of the fracture of the adjacent bone and a rupture of the
ane, then naturally bone conduction was greater than air

conduction; later when the perforation of the tympanic membrane has healed and when there has been no permanent damage to the mechanism of the middle ear conductivity, then bone conduction equals air conduction and the acuity of hearing increases, until finally within a period of months air conduction is again normally greater than bone conduction and the impairment of the infected ear can only be ascertained by most careful tests. Unfortunately, however, this happy result does not always occur, but it tends to occur in all of these patients in whom the auditory nerve itself is not damaged and where the anatomy of the middle ear has not been permanently impaired.

The profuse bleeding from the right external auditory canal through a torn right tympanic membrane as the result of the fracture of the skull, and particularly of the petrous portion of the right temporal bone, made it possible for a large amount of intracranial "free" hemorrhage to escape and thus a sort of "natural" decompression was effected, in that the intracranial pressure was thus lessened and possibly of sufficient amount to permit the absorption of the remaining intracranial hemorrhage and cerebral edema by natural means, and thus the mechanical relief by means of a subtemporal decompression and drainage was avoided. The danger of infection extending in these cases through the lacerated tympanic membrane and then intracranially through the line of fracture is to be feared, chiefly when attempts are made to "clean" the external auditory canal by "wiping" it out, syringing and other such meddlesome procedures; naturally, if the discharge of blood or of cerebrospinal fluid should continue longer than 48 hours, then the danger of an ascending infection with resulting meningitis becomes proportionally greater. The best treatment for this condition is absolute quiet in bed, vigorous catharsis and the application loosely of sterile gauze pads changed frequently, to the entire lobe of the ear; it is indeed rare for an ascending infection and meningitis to occur in these patients if the treatment as outlined above is carried out.

Lumbar punctures were not utilized therapeutically more than once for this patient because the severe headache was immediately lessened following the lumbar puncture and withdrawal of 15 c.c. of clear cerebrospinal fluid, and as the mild signs of an increased intracranial pressure subsided, a second lumbar puncture to lower still more the intracranial pressure was not considered necessary. It is unusual, however, for the severe headache of these patients to be permanently lessened by merely one lumbar puncture.

Although a large amount of blood was discharged from the right ear and presumably coming from the intracranial cavity, yet both lumbar punctures contained only clear cerebrospinal fluid so that either all of the "free" subdural or subarachnoid hemorrhage had escaped through the ear, which is very improbable, or the intracranial "free" blood had not passed downward into the spinal canal, and this latter explanation is more likely. It is possible, however, that the blood escaping from the ear did not come through the line of fracture opening into the ear but that it was due to a local injury of the tympanic membrane itself; the amount of blood, however, was rather large for a mere local injury to the ear, but as no cerebrospinal fluid was observed in the bloody discharge, it cannot be definitely stated that this blood came from the intracranial cavity.

Case 18.—Fracture of base of the skull; signs of a mild increase of the intracranial pressure. No operation. Excellent recovery.

No. 911.—Annie. Seventy years. White. Widow. Maid. Ireland.

Admitted October 19, 1917, Polyclinic Hospital.

Discharged November 13, 1917—25 days after injury.

Family history negative.

Personal history negative.

Present Illness.—Patient was hit by a trolley car and dragged by its front fender; unconscious; brought to the hospital in the ambulance.

Examination upon admission (40 minutes after injury).—Temperature, 98.2°; pulse, 92; respiration, 28; blood-pressure, 120. An old but hardy woman; unconscious. Fracture of outer third of left clavicle. Bleeding from the left ear; no cerebrospinal fluid observed. Paralysis of left side of face (peripheral type). No convulsive movements or twitchings. Pupils equal but react sluggishly. Reflexes—knee-jerks increased but equal; no Babinski; abdominal reflexes absent. Fundi—opacities of both cornea prevents an accurate examination. Lumbar puncture—cerebrospinal fluid clear and under normal pressure (9 mm.). Urine—slight trace of albumen; otherwise negative.

Treatment.—Expectant palliative; appropriate position and dressing for fracture of the clavicle.

Examination (20 hours after admission).—Temperature, 99.2°; pulse, 82; respiration, 22; blood-pressure, 138. Semiconscious but can be aroused by speaking loudly to her. Multiple bruises over body. Bleeding from left ear has ceased; otoscopic examination reveals a large laceration of posterior portion of the left drum; extensive left mastoid ecchymosis. Weakness of left side of face less marked. Pupils equal and react normally. Reflexes active and equal; no Babinski; abdominal reflexes obtained with difficulty. Fundi—retinal veins dilated slightly but no edema about the optic disks, which are rather pale. X-ray (Doctor G. W. Welton) "shows fracture of the squamous portion of the left temporal bone extending to the base and involving the left auditory canal" (Fig. 56).

Examination (7 days after admission).—Temperature, 99.4°; pulse, 74; respiration, 20; blood-pressure, 144. Excellent improvement. Patient complains of general weakness and slight frontal headache; general stiffness all over. Left mastoid ecchymosis gradually fading. Difficult to elicit weakness of left side of face by special tests. Reflexes exaggerated but equal; no Babinski; abdominal reflexes sluggish but equal. Fundi—no definite blurring of optic disk margins.

Examination at discharge (November 13, 1917—25 days after admission).—"I shall never get over the shock of the injury; I feel weak all over." No complaints of headache. No facial weakness can be elicited. Reflexes active but otherwise negative. Fundi negative. Hearing—left ear impaired—bone conduction being greater than air conduction.

Last Examination (August 21, 1918—9 months after injury).—No complaints referable to injury, still feels weak in legs and back. No paralysis of face. Reflexes negative. Fundi negative. Hearing—left ear less acute than right; bone conduction, however, only equals air conduction.

Remarks.—The paralysis of the left side of the face, which was of the peripheral type in that the left forehead muscles were involved, and the early disappearance of this facial weakness, are indicative of merely an edematous compression of the facial nerve in its bony aqueduct in the petrous portion about the middle ear—the usual site for peripheral lesions of the facial nerve; in these traumatic cases when the skull has been fractured, a complete tear of the facial nerve is always to be feared, but this unfortunate complication occurs in only a small percentage of patients in whom there is a peripheral facial paralysis, because it is usually one of temporary duration in that it results from an edematous swelling of the nerve within its bony canal. If the facial nerve, however, should be torn in these patients, and the facial paralysis may be considered permanent if no improvement has occurred within one year following the injury, then a peripheral anastomosis of one-half of the central cut end of the ipsolateral hypoglossal nerve to the distal cut end of the facial nerve as it emerges from the stylo-mastoid foramen should be considered before the atrophy of the facial muscles and their resulting contractures should become extreme; this operation of anastomosis is technically difficult but of little risk to the patient, and it causes no facial disfigurement.[1] The clear cerebrospinal fluid at lumbar puncture and yet a fracture of the skull being present is an interesting but a rather common observation.

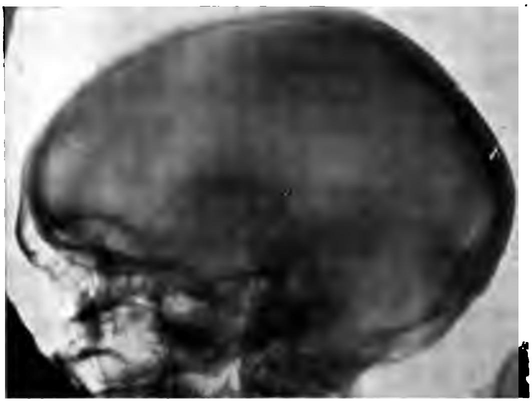


FIG. 56.—Linear fracture of left squamous bone extending into left middle ear with a rupture of the left tympanic membrane. The mild increase of intracranial pressure lessened by the expectant palliative treatment. Excellent recovery.

This patient has made an excellent recovery when you consider her age of seventy years, and yet she was in unusually good physical condition, so that she could withstand both the shock of such a severe injury and its pressure effects remarkably well; the absence of alcohol and cardio-renal disease was a most important factor in her ultimate recovery.

The otoscopic examination is a very valuable diagnostic aid in cranial injuries; although in many patients there may be no escape of blood and cerebrospinal fluid from the ear, yet the presence of free blood within the middle ear, as shown by a bluish tympanic membrane, and also possibly the presence of cerebrospinal fluid, would indicate a fracture of the adjacent bone, even in the absence of a laceration of the tympanic membrane, which usually results and thus permits the escape of blood and cerebrospinal fluid from the external auditory canal. The impairment of hearing of these patients might be lessened earlier and their ultimate recovery assured with more confidence if a paracentesis of the tympanic membrane is performed and the drainage of this blood thus made possible; the organization of this blood-clot in the middle ear must cause a definite impairment of hearing—possibly not sufficient for the patient to be conscious of it. The danger of a possible infection extending through the punctured tympanic membrane must be remembered, but the risk is so slight with proper precautions of asepsis and after-treatment that it is more a question of obtaining the greatest recovery of hearing possible rather than the fear of a possible complication of infection (unless conditions are not the best and then the paracentesis should not be performed).

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[1] The operative treatment of chronic peripheral facial paralysis. *J.A.M.A.*, May 11, 1918.

Ecchymosis of the mastoid area not due to a direct local injury is much stronger evidence of an adjacent fracture of the skull than the presence of subconjunctival hemorrhage, which occurs very frequently without a fracture of the adjacent bones and is due to a rupture of one of the many subconjunctival vessels. The absence of a fracture of the skull, and particularly of the base, merely because the X-ray pictures are negative, naturally cannot be definitely stated, and in this patient, especially when there are signs indicative of a fracture of the skull such as a definite left mastoid ecchymosis, "free" blood in the left middle ear and also free blood in the cerebrospinal fluid at lumbar puncture—these facts would tend to point to a fracture of the skull and especially the base of the skull in the region of the left petrous bone—the X-ray seldom revealing lines of fracture of the base and in this particular area.

Case 19.—Fracture of base of skull; signs of a mild increase of intracranial pressure. No operation. Excellent recovery.

No. 722.—Patrick. Forty-five years. White. Single. Kitchen-man. Ireland.

Admitted November 8, 1916, Polyclinic Hospital.

Discharged November 23, 1916—15 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While at work in the hotel, patient fell into the elevator shaft; apparently no loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (75 minutes after injury).—Temperature, 98.4°; pulse, 72; respiration, 20; blood-pressure, 142. Conscious but drowsy and stuporous. Profuse bleeding from right ear—mingled with cerebrospinal fluid; right mastoid ecchymosis. Right facial paralysis of the peripheral type. Pupils equal and react normally. Reflexes—knee-jerks active but equal; no ankle clonus nor Babinski; abdominal reflexes depressed but equal. Fundi negative. Lumbar puncture—blood-tinged cerebrospinal fluid under normal pressure (approximately 9 mm.).

Treatment.—Expectant palliative.

Examination (30 hours after admission).—Temperature, 99.8°; pulse, 74; respiration, 20; blood-pressure, 144. Perfectly conscious; complains of severe frontal and occipital headache. Scanty straw-colored discharge from right ear (approximately 4 drops per minute); right mastoid area very ecchymotic and boggy. Right facial paralysis persists. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—slight dilatation of retinal veins; lower quadrant of nasal margins of optic disks indistinct. X-ray (Doctor W. H. Stewart)—"no fracture of the skull observed."

Examination (4 days after admission).—Temperature, 99°; pulse, 76; respiration, 18; blood-pressure, 144. No complaints except for dull aching headache; "ears stopped and then my head began to ache." Straw-colored discharge from right ear has ceased; otoscopic examination reveals small laceration of the posterior lower quadrant of the right tympanic membrane; left ear negative. Right facial paralysis remains the same. Reflexes active but otherwise equal. Fundi—retinal veins enlarged; distinct edematous blurring along the nasal margins of both optic disks. Lumbar puncture—clear cerebrospinal fluid under a slightly increased pressure (approximately 11 mm.); 15 c.c. withdrawn slowly for therapeutic effect.

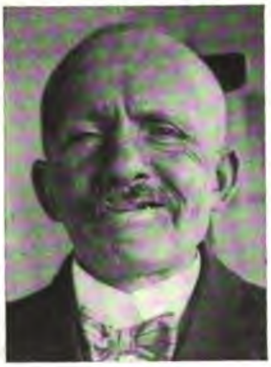

FIG. 57.—Right facial paresis following a complete right facial paralysis of the peripheral type and most probably due to an edematous compression of the right facial nerve in its bony canal adjacent to the middle ear. Entire recovery.

Treatment.—Expectant palliative continued; massage and galvanism for the right facial muscles.

Examination at discharge (15 days after admission).—Temperature, 98.6°; pulse, 70; respiration, 18; blood-pressure, 142. Complains of slight frontal headache "in the eyes"; otherwise negative. No discharge from right ear; right mastoid area still slightly discolored. Hearing impaired in right ear; bone conduction greater than air conduction. Reflexes active but otherwise negative. Fundi—retinal veins slightly enlarged but the nasal margins of the optic disks are now clear. Right facial paralysis is much improved; patient can close the right eye and wrinkle forehead slightly (Fig. 57).

Examination (September 20, 1917—10 months after injury).—"Well as ever," except for a slight dizziness when stooping. Reflexes active but otherwise negative. Fundi negative. Hearing—right ear impaired; bone conduction slightly greater than air conduction. No right facial weakness can be elicited.

Last Examination (July 29, 1918—20 months after injury).—No complaints except apparently an increasing alcoholism. Reflexes negative. Fundi negative. Hearing—right ear still impaired; bone conduction still greater than air conduction. Facial musculature normal.

Remarks.—Although the X-ray picture did not reveal a line of fracture (and it rarely does when the fracture is limited to the base of the skull, and particularly of the middle fossa), yet there must have been a fracture of the skull because cerebrospinal fluid escaped through the lacerated right tympanic membrane and emerged at the right auditory meatus.

The presence of blood in the cerebrospinal fluid is merely an indication that the cranial injury has caused "free" blood to escape into th
arachnoid and subdural spaces—particularly the former:
not true, however, because clear cerebrospinal flui

lumbar puncture in patients having a large amount of "free" hemorrhage, both subarachnoid and subdurally. A fracture of the skull may be present or not—the intracranial hemorrhage is not dependent upon whether a fracture of the skull is present or not. It would seem to be unusual, however, for an extensive fracture of the skull to occur, and especially of the base of the skull to which the dura is adherent, without causing some "free" blood to escape into the subdural or subarachnoid spaces due to a rupture of one or more small vessels; if of small amount, this bleeding is of little importance clinically, as it can be absorbed by "natural" means, and it is only when it is associated with a marked cerebral edema or when the hemorrhage itself is of large amount, that the signs of a marked increase of the intracranial pressure necessitates a mechanical relief of this intracranial pressure by means of the subtemporal decompression and drainage.

The statement and excellent observation of the patient that "ears stopped and then my head began to ache" are most interesting and undoubtedly correct, because the escape of blood and of cerebrospinal fluid from the right ear permitted a lessening of the intracranial pressure and thus no severe headache occurred, but when this exit for the cerebrospinal fluid became blocked, then the intracranial pressure increased until the patient had definite pain throughout the head as a result of this increased intracranial pressure expanding and stretching the dura. It was for this reason that a second lumbar puncture was performed and 15 c.c. of cerebrospinal fluid removed, and by thus again lessening the increased intracranial pressure, the headache was relieved for a period of almost 8 hours.

If it were not for the danger of infection extending through the lacerated tympanic membrane and then intracranially through the line of fracture with a resulting purulent meningitis, it would always be advisable for the discharge of cerebrospinal fluid through the ear to continue for a number of days until any increase of the intracranial pressure would be entirely relieved; such drainage afforded by the escape of a large amount of the cerebrospinal fluid and of the intracranial hemorrhage would obviate the necessity of cranial operations upon these patients in all but the most severe and extreme conditions. On account of this danger of infection, if the discharge of blood and particularly of cerebrospinal fluid has continued longer than 48 hours, it is frequently advisable to perform repeated lumbar punctures daily and 15–20 c.c. slowly removed each time, in order by this means of drainage to lessen the increased intracranial pressure and thus permit the opening in the line of fracture through the ear to become closed and thereby lessening the danger of infection through that channel.

The right facial paralysis of the peripheral type (as indicated by the muscles of the right half of the forehead being involved) was most probably due to an edematous compression of the right facial nerve itself in the bony aqueduct adjacent to the right tympanum: its complete recovery of function would confirm this belief. This type of edematous compression of the facial nerve is the most common form following cranial injuries. It is rare for the l nerve to be completely severed as the result of the line of fracture ugh the bony canal of the facial nerve.

Acute severe brain injury associated with mild signs of increased intracranial pressure and a possible fracture of the base of the skull. No operation. Symptoms and signs persisting for one year and then an excellent recovery.

No. 056.—Harriet. Forty-eight years. White. Married. Housework. U. S.

Admitted June 9, 1913, White Plains Hospital. Consultation June 11, 1913—2 days after injury. Referred by Doctor G. S. Amsden, White Plains.

Discharged July 18, 1913—39 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While driving an automobile, patient had a collision, throwing her from the car and striking her head against the ground; immediate loss of consciousness; taken to the hospital in the automobile where, upon admission (30 minutes later), the temperature was 100, pulse 90 and respiration 24; there was no bleeding from nose, mouth or ears; knee-jerks were much exaggerated but equal, and there was present a double Babinski.

Treatment.—Expectant palliative.

Consultation with Doctor Amsden on June 11, 1913—2 days after injury.—Temperature, 99.8°; pulse, 94; respiration, 22; blood-pressure, 146. Well-nourished and developed. Unconscious, but could be roused by firm supra-orbital pressure, although unable to reply to questions. No paralyses elicited. Right subconjunctival hemorrhage, right orbital and right mastoid ecchymoses; no clotted blood in nares or external auditory canals. Pupils equal and react normally. Reflexes—patellar exaggerated, left possibly greater than right; no ankle clonus but left Babinski; abdominal reflexes not obtained (abdominal wall being rather fat and pendulous). Fundi—retinal veins dilated; nasal halves of both optic disks blurred by edema, right possibly more than left. Lumbar puncture—blood-tinged cerebrospinal fluid under increased pressure (approximately 15 mm.).

Treatment.—The following note was made by me and attached at that time to the history of the patient: "As it seems that the condition of the patient is improving, especially the unconsciousness, which was becoming lighter—more of a comatose condition, I do not urge an immediate decompression, although I consider it advisable—not for fear the patient will die, but rather to avoid post-traumatic conditions." It was decided to await developments. Usual expectant palliative treatment.

Consultation with Doctor Amsden (June 14, 1913—5 days after injury). —Temperature, 99°; pulse, 84; respiration, 22; blood-pressure, 140. Patient is now conscious—complaining of severe headache. Marked paraphasia—sensory as well as motor. Pupils equal and react normally. Reflexes—patellar exaggerated, left possibly greater than right; no ankle clonus and the left Babinski is now easily exhausted; abdominal reflexes cannot be obtained. Fundi—retinal veins dilated; nasal halves of both optic disks blurred as at preceding examination.

Treatment.—Expectant palliative. Patient made a gradual recovery; complained of dull persistent headache. Paraphasia continued for 2 weeks; amnesia present for 20 days and then gradually improved. Patient easily fatigued and distressed by any noise. Poor memory for proper names persisted after discharge on the thirty-ninth day following injury; at that time, the reflexes were increased but equal and there was no Babinski present.

Examination (January 1, 1914—almost 7 months after injury).—A definite improvement has occurred; it is only when tired that patient complains of discomfort in the head—a sense of pressure; occasional spells of dizziness; restlessness with insomnia at times. Rarely perseveration in the use of words. Memory for proper names still impaired. No marked changes in temperament—possibly more irritable. Slight tremor, with mild ataxia, of both hands, especially the left; tendency to positive Romberg. Pupils equal and react normally. Reflexes— patellar active but equal; no ankle clonus nor Babinski. Fundi—retinal veins not abnormally dilated, although the vessel walls appear slightly thickened; slight haziness about nasal margins of both optic disks which are rather pale; both physiological cups are shallow from new tissue formation.

Examination (June 1, 1914—12 months after injury).—Patient still complains of heaviness and fulness in the head, especially upon exertion or when fatigued. No definite changes in personality; memory for recent events not so good as before the injury; slight paraphasia at times—frequently unable to use well-known words. Pupils equal and react normally. Reflexes—patellar very active but equal; no ankle clonus nor Babinski. Fundi same as at preceding examination. No impairment of vision other than a subjective blurring and haziness. The following note was made by me at this time upon the patient's history: "It will be most interesting to observe this patient for a number of years in order to ascertain the permanent impairment; she may entirely recover and yet it would be surprising if she did so; post-traumatic conditions in this type of patient are most persistent. I now feel that I should have advised a cranial decompression at my first examination more vigorously than I did; in my opinion, it would have been the safer procedure."

Examination (September 20, 1915—27 months after injury).—During the past year patient has made such a marked improvement that she may be considered as practically well; no longer has headaches, and it is rare for her to have even a feeling of fulness in the head. The memory has become more normal and there is no longer a paraphasia. In almost every way, patient is the same as before the injury. Reflexes active but otherwise negative. Fundi—retinal veins of normal size, although their walls are thickened by new tissue formation; no edematous blurring of details of optic disks; new tissue formation naturally persists.

Last Report (September 22, 1918—63 months after injury).—Husband states that patient is a well woman "in every way"; no complaints except for an occasional dull headache; no change of personality and no emotional impairment. No difficulty of speech and memory is now excellent.

Remarks.—This has been a most interesting case to follow in that at the time of the injury and during the first year following it, it appeared that the patient was not going to regain her former good health as before the injury; the persistence of the signs of a mild increase of the intracranial pressure for the period of one year following the injury made me feel at the time that the condition would probably be a permanent one. The rapid improvement mentally, emotionally and physically, following the adjustment of a slight domestic annoyance was, I believe, a definite factor in causing this marked improvement to occur.

It would seem that this patient had had a chronic cerebral edema associated with a mild intracranial hemorrhage—there being no fracture of the skull demonstrated, although it may have been present, and that the period of time necessary for the cerebral edema to be absorbed was in this patient a little over one year; a rigid and strict expectant palliative treatment was administered throughout her convalescence, and I believe that this careful medical supervision was an important factor in her ultimate recovery. In many respects the post-traumatic history of this patient is unusual, for it is most rare for patients to recover ultimately so well after their symptoms and signs have persisted for a period of one year after the injury; it is for this reason that I still feel that it is the safer procedure to perform a subtemporal decompression upon patients similarly affected as this patient, for the risk of the operation is slight compared with the great danger of the many post-traumatic complications, and even if an occasional patient, just as this patient, should make an excellent recovery even if the operation to relieve the increased intracranial pressure had not been performed, yet the risk to which the majority of these patients would be exposed (and I believe the average would be as high as 9 out of 10) is a far greater danger than the slight risk of the operation itself, and the benefit following the operation would be immediate, whereas even in the occasional fortunate patient of this type who finally recovers the former good health without an operation, the period of time necessary for the convalescence is usually the same as for this patient—an average of 12 months.

b. Doubtful recovery.

CASE 21.—Fracture of base of skull; signs of a mild increase of the intracranial pressure. No operation; repeated lumbar punctures. Doubtful recovery.

No. 181.—Matilda. Sixty-seven years. White. Single. Retired school teacher. U. S.

Admitted March 11, 1915, Polyclinic Hospital. Referred by Doctor W. A. Scruton.

Discharged April 6, 1915—26 days after injury.

Family history negative.

Personal history negative; always well and strong.

Present Illness.—On night before admission, patient fell down a flight of stairs while chasing a burglar; has been unconscious since the accident; brought to the hospital in the ambulance.

Examination upon admission (18 hours after injury).—Temperature, 99.4°; pulse, 88; respiration, 28; blood-pressure, 148. Patient could be roused to semiconsciousness at intervals; could not talk intelligibly; did not recognize surroundings; tendency to drawl, rhyme and whine what words she spoke, such as die, try, by, etc. Nothing she said had any sense, but upon being aroused the sensation of stimulation would apparently cause her to rhyme other words; the sensation of moisture following the expelling of an enema caused her to say "I am wet," and immediately after to make another sentence of no particular meaning, but of the same length, and all ending with a word rhyming with wet, as bet, let, set, etc. Well-developed and nourished for her age. Laceration of about 3½ inches long over left occipito-parietal region extending down to the bone. This had been sutured and drained by the physician who had attended patient immediately after the fall. Slight laceration of right orbital region and marked ecchymoses about both eyes; left eye nearly closed. Clotted blood in left auditory canal; marked left mastoid ecchymosis; otoscopic examination revealed a laceration of the left ear drum. Lumbar puncture—cerebrospinal fluid clear and under a slightly increased pressure (approximately 11 mm.). X-ray (Doctor A. J. Quimby)—"no line of fracture to be observed." Pupils equal but react sluggishly. No facial paralysis. Reflexes—knee-jerks equal and not much exaggerated; no Oppenheim, Gordon, Babinski or ankle clonus; abdominal reflexes not obtained. Eye grounds show no appreciable blurring; retinal vessels of normal size.

Treatment.—Expectant palliative.

Examination (7 days after admission).—Temperature, 99°; pulse, 82; respiration, 26; blood-pressure, 146. Patient has remained in a dazed condition during past week; very delirious at times; refused all medications and screamed loudly at all attempts to aid her. At end of this time she improved somewhat mentally; recognized her brother but could not talk coherently; cried and screamed for no apparent reason and talked nonsense continually. Reflexes active but otherwise negative. Fundi—retinal veins enlarged; otherwise negative. Repeated lumbar punctures upon 5 successive days allowed each time 20 c.c. of clear cerebrospinal fluid to drain off under slightly increased pressure; the patient became quieter and would even sleep within an hour after each puncture. During the following week the improvement was not so marked; laceration had entirely healed *per primam*, but she complained of pain in that area and general headache; could read and at times talk fairly sensibly, but soon would run off into delirious chatter. Physical examination was practically negative; reflexes active but otherwise negative. Fundi—retinal veins slightly enlarged but otherwise negative. Lumbar puncture (15 days after admission)—cerebrospinal fluid clear but ran out under a slightly increased pressure (approximately 11 mm.).

Examination at discharge (25 days after admission).—Temperature, 98.8°; pulse, 80; respiration, 24; blood-pressure, 146. Much better in every way; clearer mentally but still confused, repeating, "I must go to the school to my pupils" and "I am old enough to be your mother and you must let me go to the school." (Patient has not taught school for 6 years.) Perseveration of words is not present nor any motor or sensory aphasia, but a sort of "senile chatter" and a childishness which were not present before the head injury. Reflexes active but not abnormal. Fundi negative.

Examination (August 24, 1917—28 months after injury).—Patient has come to me repeatedly for examinations during the past 2 years; very much improved mentally and emotionally, although never having as normal a mental condition as before the injury. Has become very irritable and "very difficult to live with." Reflexes negative. Fundi negative.

Last Report (December 12, 1919—56 months after injury).—Patient resides in a "home for old and indigent teachers." "Possibly more irritable but otherwise very much as before the injury."

Remarks.—It would seem in this patient that the head injury produced an acute condition of "wet" brain so that this condition, associated with her age, produced the mental and emotional instability. It will be very instructive and important to obtain a post-mortem examination later.

The clotted blood in the left auditory canal associated with a laceration of the left tympanic membrane (as revealed by otoscopic examinations), and the distinct ecchymosis of the left mastoid area would indicate a fracture of the base of skull in the region of the petrous portion of the left temporal bone; the absence of blood in the cerebrospinal fluid and the negative röntgenogram would not exclude a fracture of the skull from being present in this area of the base of the skull.

It was interesting to observe the temporary periods of relief, both of the mild delirium and general restlessness, following each of the lumbar punctures and removal of 20 c.c. of cerebrospinal fluid; the patient would almost immediately become quiet and within one-half hour gradually fall asleep; this restful period might continue for 4 to 8 hours, when upon awakening the patient would again become noisy and require restraint. This persistent cerebral edema was undoubtedly the cause of her mental and emotional instability to which she was more susceptible on account of her age.

CASE 22.—Fracture of base of skull; signs of a mild increase of intracranial pressure. No operation; repeated lumbar punctures. Doubtful recovery.

No. 170.—Robert. Forty-two years. White. Married. Laborer. Ireland.

Admitted August 26, 1915, Polyclinic Hospital. Referred by Doctor Alexander Lyle.

Discharged September 12, 1915—16 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While crossing the street, patient was hit by an automobile; unconscious; brought to the hospital in the ambulance.

Examination upon admission (40 minutes after injury).—Temperature, 97.8°; pulse, 84; respiration, 24; blood-pressure, 132. Well-developed and nourished; unconscious and in shock. Bleeding profusely from nose and both ears; no mastoid ecchymosis. Pupils equally dilated and react to light sluggishly. Reflexes difficult to elicit, but no ankle clonus nor Babinski; abdominal reflexes absent. Fundi negative.

Treatment.—Expectant palliative.

Examination (10 hours after admission).—Temperature, 99.8°; pulse, 72; respiration, 18; blood-pressure, 142. Unconscious; respiration rather labored. Nasal bleeding has stopped but cerebrospinal fluid continues to leak from both ears—the left more profusely; marked left mastoid ecchymosis. Pupils equal and react normally. Reflexes less difficult to elicit; equal; no Babinski; abdominal reflexes not obtained. Fundi—retinal veins dilated; nasal margins indistinct and blurred. Lumbar puncture—blood-tinged cerebrospinal fluid under an increased pressure of approximately 12 mm. X-ray (Doctor A. J. Quimby)—"no line of fracture can be seen."

Treatment.—Expectant palliative. Repeated lumbar punctures were performed daily for 4 days, when patient became conscious; at each puncture 15–20 c.c. of blood-tinged cerebrospinal fluid were slowly and carefully removed; pressure at the end of the puncture would frequently be less than one-half of the pressure before the puncture.

Examination (6 days after admission).—Temperature, 100°; pulse, 70; respiration, 18; blood-pressure, 144. Conscious and complains of severe general headache. No blood nor cerebrospinal fluid escaping from the ears; otoscopic examination reveals a bilateral laceration of both tympanic membranes. Reflexes markedly exaggerated but equal; no Babinski; abdominal reflexes present and equal. Fundi—considerable dilatation of the retinal veins; edematous blurring of the nasal margins of the optic disks.

Examination at discharge (16 days after admission).—Temperature, 98.6°; pulse, 76; respiration, 20; blood-pressure, 146. Conscious and no complaints, except for a dull throbbing headache each morning; by noontime, the headache has gone. Hearing impaired and referable to double middle ear involvement, bone conduction being greater than air conduction. Reflexes active but otherwise negative. Fundi—retinal veins still enlarged and the nasal margins of both optic disks are not yet distinct.

Examination (February 12, 1917—18 months after injury).—Patient has not worked since the injury owing to almost daily headaches, early fatigue and general lassitude: sleeps heavily and for 14 hours at a time. Hearing is impaired but is better than 3 months ago. Reflexes active but otherwise negative. Fundi—slight dilatation of the retinal veins and also a slight but distinct blurring of the nasal margins of the optic disks. Lumbar puncture—clear cerebrospinal fluid under a pressure of approximately 10 mm.

Treatment.—Light diet; daily catharsis; light work requiring very little physical exertion, but sufficient to keep patient's mind upon his work and thus tend to forget himself and troubles.

Last Examination (August 23, 1918—36 months after injury).—Still complains of headache though not so severe as formerly; is "working" as a night watchman in an office. Reflexes active but otherwise negative. Fundi—retinal veins slightly dilated; nasal margins practically clear and free of edematous blurring. Patient refused a lumbar puncture to lessen the pressure of the cerebrospinal fluid.

Remarks.—It was mistaken judgment not to have advised and to have performed a subtemporal decompression and drainage upon this patient—not only to obtain a recovery of life (which was possible with merely the expectant palliative treatment), but to secure for the patient his former good health as much and as early as possible. I do not consider that the repeated lumbar punctures were of sufficient relief to the increased intracranial pressure in that the signs of pressure quickly returned and have continued in a mild degree up to the present time; as a result of this increased pressure, he has become definitely impaired, both mentally and emotionally, and is no longer capable of performing a real day's work. At the time of the injury, it was believed that the bilateral drainage of blood and cerebrospinal fluid through both ears (due to a fracture through the middle fossa) would lessen sufficiently the increased intracranial pressure, and thus, not only make an operation of decompression unnecessary but secure a normal individual ultimately; unfortunately, this opinion was mistaken and it would undoubtedly have been better judgment to have performed the operation of subtemporal decompression and drainage as soon as the initial shock of the injury had disappeared.

Case 23.—Fracture of base of skull; signs of a mild increase of intracranial pressure. No operation. Doubtful recovery.

No. 899.—Urling. Fifteen years. White. School. U. S.

Examined in consultation with Doctor George D. Stewart—June 15, 1917, 12 days after injury.

Family history negative.

Personal history negative, except for chorea 2 years ago.

Present Illness.—Twelve days ago (June 3, 1917), while riding a horse, patient was violently thrown to the ground, striking her head; immediate loss of consciousness. No bleeding from the nose, mouth or ears; no mastoid ecchymoses. Carried to her home in severe shock, which she survived after several days, but has not regained consciousness; she has, however, become restless during the past 3 days and is now able to swallow liquids. Right hemiplegia present. Lumbar puncture, performed by Doctor Stewart on the second day after the injury, revealed the cerebrospinal fluid blood-tinged and not under abnormal pressure.

Examination (June 15, 1917—12 days after injury).—Temperature, 99.4°; pulse, 86; respiration, 26; blood-pressure, 132. Well-nourished; unconscious though supra-orbital pressure causes patient to struggle and moan; she does not open her eyes, however. Definite paralysis of entire right side of body—right side of face (the right frontal muscles being but slightly, if at all, involved and therefore the paralysis is a cortical one) and the right arm and the right leg; patient moves left side freely, but no attempt made to move the right side; definite loss of speech (patient being right-handed and also parents and grandparents), therefore her motor speech centre is in the left hemisphere—the side of the brain involvement causing the right hemiplegia. Pupils—left larger and both pupils react to light sluggishly. Reflexes: knee-jerks—right greater than left; right ankle clonus and right Babinski, Gordon and Oppenheim; right abdominal reflexes distinctly less active than left. Fundi—slight dilatation of retinal veins; distinct edematous blurring along the nasal margins of both optic disks. Lumbar puncture shows straw-colored cerebrospinal fluid not under a greatly increased pressure (no mercurial manometer was at hand and the cerebrospinal fluid was merely allowed to drop out of the puncture needle—a crude and very inaccurate method of estimating the pressure of the cerebrospinal fluid).

Treatment.—Expectant palliative treatment was continued in the belief that the condition was one of brain laceration and not under increased pressure—therefore, no operation advisable; that is, the damage to the brain causing the hemiplegia had already occurred by a laceration of brain tissue, and since no marked intracranial pressure was present there was nothing to be done except the expectant palliative treatment.

Examination (June 22, 1917—19 days after injury).—Temperature, 98.8°; pulse, 82; respiration, 24. Patient has partially recovered consciousness, but is extremely drowsy—does not recognize relatives, unable to talk. Right hemiplegia less marked—patient now able to move right arm and right leg slightly and very awkwardly; no definite impairment of sensation. Pupils—left still larger than right and reacts sluggishly to light. Reflexes—patellar, right exaggerated; distinct right patellar and right angle clonus; right Babinski, Gordon and Oppenheim; abdominal reflexes—right depressed. Fundi—retinal veins enlarged; indefinite edematous blurring of nasal margins of both optic disks.

Treatment.—Expectant palliative continued; general massage to right side of body; passive exercise. Patient did not become entirely conscious until 33 days after injury and patient did not speak until the 42nd day following injury. Unable to walk alone until October, 1917—4 months after injury.

Examination (May 16, 1918—11 months after injury).—Patient has made a remarkably good recovery, although there is still present a definite weakness of the entire right side of body; the facial weakness can only be elicited by special tests, while there is only a very slight lameness of the right leg; right hand-grasp almost as strong as left. Slight mental retardation but no marked emotional instability. No impairment of speech can be elicited by special test phrases. (Patient's relatives are all right-handed.) Pupils equal and react normally. Reflexes: patellar—right greater than left; exhaustible right ankle clonus but right Babinski still present; abdominal reflexes—right less active than left. Fundi—retinal veins possibly slightly enlarged; lower nasal quadrant of nasal margins of optic disks obscured and hazy from edema.

Treatment.—Daily massage, both active and passive exercises; non-stimulating diet—no meat, meat-soup, tea, coffee or alcohol.

Last Examination (March 13, 1919—21 months after injury).—Patient has continued to improve in every way so that now, from a superficial examination, she might be considered a normal girl. Mentality, however, is slightly retarded in that she is very deliberate in her answers and lacks a certain mental alertness; no emotional impairment. No complaints whatever and feels "perfectly well." Pupils equal and react normally. Reflexes: patellar—right more active than left; suggestive right ankle clonus and right Babinski is possibly less vigorous than at last examination; abdominal reflexes—right less active than left. Fundi—retinal veins possibly larger than normal; lower nasal margins of both optic disks slightly blurred. X-ray (Doctor G. W. Welton)—"an irregular linear fracture of the right half of the occipital bone" (Fig. 58).

Remarks.—It is a question whether a subtemporal decompression and drainage would have ultimately benefited this patient more than the treatment as administered. It would seem that this patient had had a definite cerebral laceration of the left motor cortex sufficient to cause a right hemiplegia associated with a motor aphasia, but no impairment of sensation;

it may have resulted from a subcortical lesion but it would be most unusual for the lesion to have been situated in the internal capsule and yet no impairment of sensation resulting. The mild signs of an increased intracranial pressure could have been "taken care of" by the natural means of absorption, and this patient would have made an excellent ultimate recovery if the damage and laceration to the cerebral tissue had not occurred. Naturally, the great danger of epileptiform seizures occurring later in the life of this patient is a serious one—so much so that her life should be restricted and limited to simple pleasures, a non-stimulating diet and the best of hygiene.

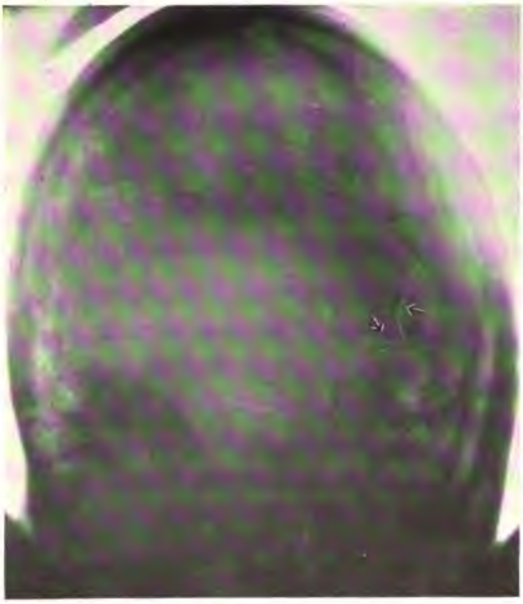

FIG. 58.—An irregular linear fracture of right half of occipital bone. A definite increase of the intracranial pressure treated by the expectant palliative method. Doubtful recovery.

The situation of the fracture as revealed by the röntgenogram is a most dangerous one, so that the patient was indeed fortunate to have recovered life itself; these subtentorial fractures of the skull radiating downward to the foramen magnum frequently produce a direct medullary compression by edema or hemorrhage, or by their extension into the lateral or sigmoid sinus and thus causing a large subtentorial hemorrhage to occur—and the early death of the patient from direct medullary compression. These patients rarely survive a period of time sufficient for an operative procedure, and even if they should live longer than several hours, the operation of suboccipital decompression and drainage is a much more formidable procedure than the simple subtemporal method. Most of the autopsies in my series of patients show a subtentorial fracture or a subtentorial hemorrhage and edema.

B. *Marked increase of intracranial pressure.*

a. Excellent recovery.

CASE 24.—Fracture of base of skull; marked increase of intracranial pressure. No operation. Excellent recovery.

No. 25.—Harry. Seventeen years. White. Elevator boy. U. S.

Admitted March 29, 1914, Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Discharged April 12, 1914—14 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While walking in his sleep, patient fell out of a fourth-story window into back yard; it is believed that a clothes line obstructed his fall; unconscious; brought to the hospital in the ambulance.

Examination upon admission (40 minutes after injury).—Temperature, 99.6°; pulse, 80; respiration, 24; blood-pressure, 120. Well-developed and nourished; semiconscious. Left Colles fracture. Contusion, hematoma and laceration on right side of head with a definite tender point over right temple. Bleeding from both ears and nose; bilateral mastoid ecchymoses. Pupils moderately dilated but equal; normal reaction. Reflexes—knee-jerks active—left greater than right; left Babinski; abdominal reflexes—right greater than left. Fundi—dilated retinal vessels; blurring of nasal margins but not of entire halves of nasal portions of both optic disks.

Treatment.—Expectant palliative; frequent examinations of fundi.

Examination (14 hours after admission).—Temperature, 100.2°; pulse, 84; respiration, 24; blood-pressure, 126. Stuporous and drowsy; remembers nothing of past night, but mind is clear on all other points. Serosanguinous discharge from both ears, and especially from the left ear. Reflexes—left greater than right; no Babinski; abdominal reflexes—left depressed. Fundi—retinal vessels dilated; edema of nasal margins persists. Lumbar puncture—cerebrospinal fluid blood-tinged and under a definitely increased pressure of approximately 13 mm. X-ray (Doctor A. J. Quimby) —"no signs of fracture."

Treatment.—Expectant palliative continued.

Examination (48 hours after admission).—Temperature, 99.4°; pulse, 78; respiration, 20; blood-pressure, 126. Conscious; complains of dull headache. Aural discharge has ceased; otoscopic examination reveals a laceration of both tympanic membranes. Hearing definitely lessened in both ears —bone conduction being greater than air conduction. Reflexes—left greater than right; no Babinski; left abdominal reflex depressed. Fundi—retinal veins slightly dilated; edema along the nasal margins less distinct.

Treatment.—Expectant palliative continued.

Examination at discharge (14 days after admission).—Temperature, 99°; pulse, 80; respiration, 22; blood-pressure, 130. No complaints, except general weakness. Pupils equal and react normally. Reflexes increased but equal; otherwise negative. Fundi—retinal veins slightly enlarged; indefinite blurring of nasal margins of both optic disks.

Examination (June 4, 1914—2 months after injury).—"All well"; no headache; wants permission to play baseball. Reflexes active but otherwise negative. Fundi negative. Otoscopic examination reveals a fibrous closure of the laceration of both ear drums. Hearing impaired—bone conduction being greater than air conduction.

Examination (September 12, 1916—30 months after injury).—No complaints. Reflexes negative. Fundi negative. Hearing of both ears less impaired; bone conduction, however, is still greater than air conduction.

Last Examination (August 10, 1918—52 months after injury).—No complaints. Reflexes negative. Fundi negative. Hearing of both ears impaired but less so—especially left; bone conduction almost equals air conduction in left ear.

Remarks.—This case illustrates the value of the expectant palliative treatment of patients having even definitely increased intracranial pressure, especially when that pressure is due to a simple edema or increase in the amount of intracranial cerebrospinal fluid; especially is this true in patients under 30 years of age. If these marked signs of increased intracranial pressure do not disappear within 48 to 60 hours, then an operative relief of that pressure is to be considered. Although in this particular patient it was better judgment not to have performed a subtemporal decompression and drainage, if this condition had occurred in a patient of middle age or even older, then it would have been distinctly dangerous to have used the expectant palliative treatment alone; possibly repeated lumbar punctures would be sufficient, but if the patient should continue to exhibit marked signs of an increased intracranial pressure for a period longer than 4 days, then a mechanical relief of this increased pressure by means of a subtemporal decompression and drainage would be advisable—being the safer procedure, not only as to the immediate recovery of life, but particularly an ultimate recovery of the former normal condition and good health.

The profuse discharge of blood from both ears, associated with a laceration of both tympanic membranes and an extensive ecchymosis of both mastoid areas—these signs indicate a fracture of the middle fossa of the base of the skull; the X-ray being "negative," is the usual report for such fractures of the base. Undoubtedly this escape of a large amount of intracranial "free" blood prevented the intracranial pressure from becoming severe and thus it was possible to avoid a cranial operation—the patient having in this manner really "decompressed" himself. If an infection of the middle ear and a possible meningitis in these patients do not occur, then this complication of profuse bleeding from the ears is a very fortunate one.

Case 25.—Fracture of base of skull; a marked increase of intracranial pressure. No operation. Excellent recovery.

No. 666.—Arthur. Twelve years. White. School. England.

Admitted September 26, 1916, Polyclinic Hospital.

Discharged October 8, 1916—12 days after injury.

Family history negative.

Personal History.—Diphtheria five years ago; otherwise always well and strong.

Present Illness.—While playing upon a one-story fire-escape, patient fell backward, landing upon back of head on concrete pavement; unconscious; brought to the hospital in the ambulance.

Examination upon admission (45 minutes after injury).—Temperature, 98.2°; pulse, 92; respiration, 28; blood-pressure, 108. Well-developed boy; semiconscious and talking at random. While the examination was being made, patient had 2 slight general convulsions, each lasting 20 seconds; no localizing signs. Large hematoma over left occipito-parietal region. Profuse bleeding from both ears; definite mastoid ecchymosis and tenderness. Pupils equal and react normally. Reflexes—knee-jerks exaggerated but equal; double Babinski and double exhaustible ankle clonus; abdominal reflexes absent. Fundi (Doctor J. A. Kearney)—"surface of disks brick red; nasal margins of disks slightly blurred. Vessels are normal."

Treatment.—Expectant palliative; vigorous treatment of shock.

Examination (10 hours after admission).—Temperature, 99.6°; pulse, 88; respiration, 26; blood-pressure, 118. Conscious but very restless and irritable (requiring codeine). Left parietal hematoma, size of small egg and very tense. Bleeding from ears has stopped; otoscopic examination reveals a laceration of both tympanic membranes. Reflexes active but otherwise negative; no ankle clonus nor Babinski; abdominal reflexes are present and equal. Fundi (Doctor J. A. Kearney)—"retinal veins are now enlarged; nasal margins of both optic disks are distinctly blurred; entire retinæ, however, congested and of a brick red color." Lumbar puncture—cerebrospinal fluid blood-tinged and under an increased pressure (approximately 13 mm.). X-ray (Doctor W. H. Stewart)—"no fracture shown."

Treatment.—Expectant palliative.

Examination at discharge (12 days after admission).—Temperature, 99.8°; pulse, 82; respiration, 22; blood-pressure, 120. No complaints except "light-headed" at times. No signs of the hematoma, but both mastoid areas are slightly discolored. Reflexes active but otherwise negative. Fundi (Doctor J. A. Kearney)—"retinal vessels slightly enlarged, but no blurring edema about the disk margins." Hearing of both ears impaired—bone conduction being greater than air conduction.

Examination (April 24, 1917—7 months after injury).—No complaints; going to school daily. Reflexes active but otherwise negative. Fundi negative. Hearing only slightly impaired; otoscopic examination reveals both tympanic membranes almost normal in appearance.

Last Examination (September 10, 1918—24 months after injury).—No complaints. Hearing of normal acuity; air conduction is greater than bone conduction. Otoscopic examination negative. Reflexes active but otherwise negative. Fundi negative.

Remarks.—The rapid improvement of the hearing in this patient is very striking; even in adults similar patients recover almost normal acuity of hearing sometimes within one year, but in children particularly, the impairment of hearing due to a hemorrhage into the middle ear and a rupture of the tympanic membrane, frequently disappears within one year after the injury.

It would seem that this patient had possibly "decompressed" himself by the profuse discharge of intracranial "free" blood through the line of fracture extending from the middle fossa into both middle ears; if no infection occurs, this is an ideal way of lessening the intracranial pressure.

The variability of the superficial and deep reflexes is well illustrated in this patient and is typical of many patients having cranial injuries. There may be present inexhaustible ankle clonus and typical Babinski reflexes and yet at a second examination one hour later, it may be very difficult to elicit either, and frequently entirely impossible to do so; whether this is due to varying conditions of cerebral circulation and cerebral edema is not definitely known. In all of these patients the entire clinical symptomatology and signs must be considered together, and not too much importance placed upon individual variations unless these latter are confirmed repeatedly upon successive examinations over a period of time.

CASE 26.—Fracture of base of skull; marked signs of increased intracranial pressure. No operation. Excellent recovery.

No. 068.—Frank. Twenty years. White. Single. Student. U. S.

Admitted September 27, 1913, Polyclinic Hospital. Referred by Doctor M. Allen Starr.

Discharged October 4, 1913—8 days after injury.

Family history negative.

Personal history negative; no history of fainting spells.

Present Illness.—During the evening, the patient had been indulging in all sorts of food and non-alcoholic drinks; while riding upon an open Sixth Avenue car, he suddenly felt faint, arose, and then fell headlong to the street; loss of consciousness for several minutes; brought to the hospital in the ambulance.

Examination upon admission (30 minutes after injury).—Temperature, 100°; pulse, 98; respiration, 28; blood-pressure, 120. Well-nourished and developed; semiconscious; vomiting profusely undigested food. No bleeding from nose, mouth or ears; right mastoid ecchymosis. Pupils equal and react normally. Reflexes all increased, but no inequality; no Babinski; abdominal reflexes present and equal. Fundi—vessels slightly dilated; no blurring of nasal borders of optic disks. Nystagmus (rotary) to both right and left. Possibly a slight weakness of right half of face. Otoscopic examination reveals a hemorrhage into right middle ear, tympanic membrane, however, remaining intact.

Treatment.—Expectant palliative.

Examination (16 hours after admission).—Temperature, 99.4°; pulse, 88; respiration, 24; blood-pressure, 128. Conscious; complains of severe headache over right side of head. Doctor John Page punctured right ear drum, allowing clotted blood and a small amount of cerebrospinal fluid to escape; sense of pressure over right side of head relieved, nystagmus disappeared within 3 hours and it was, therefore, undoubtedly of vestibular origin. Reflexes increased but otherwise negative. Fundi—enlargement of retinal veins with a distinct edematous blurring of the nasal margins of both optic disks. Lumbar puncture—cerebrospinal fluid clear and under a pressure of approximately 12 mm. X-ray (Doctor A. J. Quimby)—"no fracture observed."

Treatment.—Expectant palliative continued.

Examination at discharge (8 days after admission).—No complaints, only slight unsteadiness of both legs—cannot stand upon one leg. No nystagmus. No facial paralysis. Hearing of right ear impaired; bone conduction greater than air conduction. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—retinal veins slightly dilated but margins of both optic disks are clear.

Examination (May 15, 1914—8 months after injury).—No complaints. Reflexes active but not abnormal. Fundi negative. Still some impairment of hearing of right ear and referred to the middle ear—bone conduction being greater than air conduction; otoscopic examination reveals small irregular scar on right tympanic membrane.

Examination (October 10, 1916—37 months after injury).—No complaints except for slight impairment of hearing of right ear; otoscopic examination negative. Reflexes active but otherwise negative. Fundi negative.

Last Report (September 10, 1918).—Patient is with the American Expeditionary Force in France. No complaints.

Remarks.—The impairment of hearing of the right ear is undoubtedly due to the new tissue formation in the middle ear, resulting from the organization of the former blood; the drum was punctured after the injury in order to allow this blood clot to escape and therefore to lessen the amount of new tissue formation. In the presence of a fracture of the petrous bone, the procedure is associated with a certain amount of danger for fear of infection through the incision of the tympanic membrane, and yet if the external auditory canal is well cleansed before the drum membrane is punctured, then this complication is very rare. Naturally, if the drum membrane has been torn at the time of the fracture, then no attempt should be made to cleanse the canal itself, as this procedure increases the danger of introducing infection into the middle ear; in these patients, it usually suffices to cover the ear-lobe with a sterile gauze pad loosely, so that there will be no blockage of the canal itself. The danger of infection is greater through the fracture of the cribriform plate associated with nasal bleeding and a discharge of cerebrospinal fluid than in a fracture producing bleeding through the ear. The right facial paresis was undoubtedly due to a slight edema of the facial nerve in the aqueduct of Fallopius adjacent to the middle ear; in rare cases, the bony wall separating the facial nerve and the middle ear is so thin or even absent that middle ear lesions easily compress the facial nerve.

b. Doubtful recovery.

Case 27.—Fracture of base of skull; marked signs of an increased intracranial pressure. No operation—refused. Doubtful recovery.

No. 095.—Daniel. Twenty-eight years. White. Married. Conductor. Ireland.

Admitted December 16, 1913, Polyclinic Hospital. Referred by Doctor John A. Bodine.

Discharged December 24, 1913—8 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While alighting from his trolley car, patient was struck by an automobile; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (25 minutes after injury).—Temperature, 98.2°; pulse, 94; respiration, 18; blood-pressure, 126. Well-developed and nourished. Unconscious. Laceration of scalp over left parietal area. No bleeding from nose, mouth or ears; left mastoid ecchymosis. Pupils—right larger than left; reaction to light normal. Reflexes cannot be obtained (due undoubtedly to shock); suggestive right Babinski, however. Fundi negative.

Treatment.—Expectant palliative; vigorous shock measures instituted.

Examination (48 hours after admission).—Temperature, 98.8°; pulse, 58; respiration, 18; blood-pressure, 144. Conscious but very stuporous; restless and complains of head pains. Pulse had dropped to 58 during preceding 12 hours. Pupils equal and react normally. Reflexes—knee-jerks active, right more than left; no ankle clonus but suggestive right Babinski; abdominal reflexes present and equal. Fundi—retinal veins dilated; nasal halves of both optic disks obscured by edema. Lumbar puncture—cerebrospinal fluid blood-tinged and under increased pressure (approximately 14 mm.); 10 c.c. carefully and slowly removed. X-ray (Doctor A. J. Quimby)—"indefinite oblique fracture of left squamous bone" (Fig. 59).

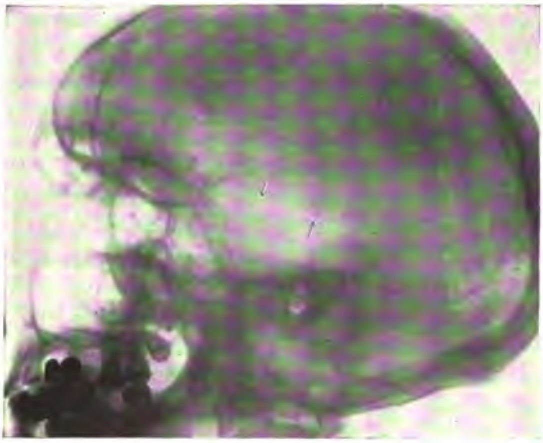

FIG. 59.—Oblique linear fracture of left squamous bone associated with a left mastoid ecchymosis. A definite increase of the intracranial pressure treated by the expectant palliative method. Doubtful recovery.

Treatment.—The operation of left subtemporal decompression advised to lessen the intracranial pressure and to facilitate the drainage and absorption of the subdural hemorrhage; the operation was refused both by the relatives and by the patient himself.

Examination at discharge (8 days after admission—patient refusing to remain in hospital longer).—Temperature, 99°; pulse, 60; respiration, 18; blood-pressure, 148. Chief complaint is headache and "I want to go home and get out of here." Scalp laceration healing *per primam*, and therefore relatives considered him "practically a well man, needing only a little rest." Ecchymosis over left mastoid area still present; otoscopic examination reveals both tympanic membranes negative. No paralysis. Pupils equal and react normally. Reflexes all active, right greater than left; suggestive right Babinski; abdominal reflexes present and equal. Fundi—retinal veins dilated; nasal margins of both optic disks obscured.

Treatment.—Relatives and patient advised regarding diet, catharsis and quiet, and no work.

Examination (January 3, 1914—17 days after injury).—Patient has resumed work; complains of slight headache and "all in all" feels fairly well. Laceration of scalp has healed. Pupils equal and react normally. Reflexes still active, right greater than left; suggestive right Babinski still persists. Fundi—edematous blurring still present about the nasal margins of both optic disks.

Examination (January 14, 1914—32 days after injury).—Patient unable to work on account of severe headache and nausea at times; dizzy spells; desires an operation now (an operation was not advised at this time owing to the belief then held that the damage had already been done and that an operation would not be of any benefit to him at this late date; we now know, however, and believe that a decompression operation at this time would have given him a definite chance of ultimate recovery). Pupils equal and react normally. Reflexes—knee-jerks exaggerated, right greater than left; suggestive right Babinski but no ankle clonus; abdominal reflexes present and equal. Fundi—retinal veins enlarged; obscuration and haziness of the nasal margins of both optic disks.

Examination (June 5, 1914—6 months after injury).—Patient complains of daily headaches and inability to work. Pupils equal and react normally. Reflexes—right more active than left; no ankle clonus nor Babinski. Fundi—nasal margins of optic disks indistinct and blurred; retinal veins enlarged; physiological cup shallow from new tissue formation. Patient refused to enter hospital for lumbar puncture.

Examination (December 2, 1916—36 months after injury).—Patient has been referred to me by a social service organization regarding his inability to work—patient just having been discharged from Blackwell's Island, where he has been confined for being a "loafer, common nuisance and vagrant" during the past 4 months; alcoholism was also a factor. Patient has become enfeebled mentally and doesn't seem to realize his pitiful condition; says "I'm all right, nothing worries me." Pupils equal and react normally. Reflexes active but apparently equal; no ankle clonus nor Babinski. Fundi—general retinal congestion and suffusion, including optic disks; retinal vessels all enlarged. Urine examination—heavy trace of albumen with numerous hyaline and granular casts.

Treatment.—Institutional care advised.

Last Report (Sept. 10, 1917—45 months after injury).—Letter from relatives states patient died at Central Islip Insane Hospital two weeks ago; that "he was never the same after the injury, took to drink and lost his mind."

Remarks.—This is a most unfortunate case—and one which occurs only too frequently following the improper treatment of brain injuries. This patient had all the signs—and yet not severe—of increased intracranial pressure, and to a degree making most advisable the mechanical relief of the increased intracranial pressure by means of a subtemporal decompression and drainage, and yet it could not be performed and the patient thus lost a chance of ultimate recovery, merely because the relatives and the patient himself considered the injury as a trivial one. No doubt alcohol was a big factor in hastening the mental, emotional and physical deterioration of this patient, but it would seem very probable that the cranial injury with its resulting cerebral impairment not only weakened this patient's emotional control but rendered him more susceptible to the deleterious effects of alcohol.

If a röntgenogram had not been made of this patient, it would still have been possible to state that a possible fracture in the region of the left temporal bone was present, chiefly on account of the left mastoid ecchymosis; the absence, however, of a laceration of the left tympanic membrane or other left middle ear involvement would have made the diagnosis of fracture of the skull very doubtful, and its presence being confirmed by the X-ray makes possible the definite diagnosis of a fracture of the skull. Bloody cerebrospinal fluid does not by any means presuppose a fracture of the skull, and as it is now well known the diagnosis and treatment of brain injuries are in no way dependent upon the presence or not of a fracture of the skull; the most severe cases of brain injuries frequently are not associated with a cranial fracture, and in many cases the presence of a fracture is of benefit to the patient in aiding him to lessen the increased intracranial pressure and thereby render unnecessary the operation of cranial decompression and drainage.

CASE 28.—Acute fracture of base of skull; marked signs of an increased intracranial pressure. No operation (refused). Doubtful recovery.

No. 85.—Charles. Twenty-eight years. White. Single. Waiter. U. S.

Admitted July 21, 1914, Polyclinic Hospital. Referred by Doctor W. S. Bainbridge.

Discharged August 20, 1914—29 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While asleep, patient fell out of a fourth-story window, striking upon head in the back-yard (earth); immediate unconsciousness; brought to the hospital in a cab.

Examination upon admission (2 hours after injury).—Temperature, 97.6°; pulse, 140; respiration, 30; (blood-pressure not obtained). Well-developed and nourished. Unconscious and in profound shock; cold clammy skin. Fracture of right tibia. Contusion and laceration over left forehead and hematoma of left eyelid. Pupils moderately dilated and react sluggishly. Reflexes—knee-jerks increased, right more than left; suggestive right Babinski; abdominal reflexes not obtained. Fundi negative.

Treatment.—Expectant palliative; shock measures instituted.

Examination (30 hours after admission).—Temperature, 99.2°; pulse, 136; respiration, 28; blood-pressure, 114. (Pulse remained 120–150 for three days; gradually regained consciousness on the third day.) General condition better, but still in severe shock. No further examination made.

Treatment.—Expectant palliative; shock.

Examination (4 days after admission).—Temperature, 99.8°; pulse, 92; respiration, 24; blood-pressure, 122. Semiconscious and restless. Clotted blood in the left auditory canal; otoscopic examination reveals a ruptured left tympanic membrane. Left mastoid ecchymosis, also ecchymosis of right mastoid and left orbit. Pupils equal and react normally. Reflexes all active but otherwise negative. Fundi—nasal margins of both optic disks blurred—especially left disk. Lumbar puncture—cerebrospinal fluid straw-colored. Laboratory report (Doctor F. M. Jeffries)—"many red blood cells"—and under an increased pressure of approximately 14 mm. X-ray (Doctor A. J. Quimby)—"indistinct line of fracture of squamous portions of both temporal bones" (Fig. 60).

Treatment.—A left subtemporal decompression advised in the belief that a lessening of the intracranial pressure and drainage of the blood and edema would be a safer procedure than the expectant palliative treatment alone. Operation refused. Expectant palliative treatment continued.

have also adopted it; it is only by following such patients over a period of months and even years, as in this series of cases, that we are impressed by the fallacy of such an attitude—in fact, it would seem that for some of these patients, and particularly for their relatives, it would have been more fortunate if the more extreme patients had died at the time of the injury, and it is my opinion that two-thirds of the patients having clinical histories in the hospitals similar to the ones in this series, never recover their former good health and normality.

The continuance of the severe shock in this case is unusual; it was only by means of most vigorous measures that this patient finally survived this extreme condition: continuous external warmth to the entire body by means of heated blankets, six hot water bags, hot black coffee and saline enemata and rectal irrigation alternately; compression bandaging of both legs was used immediately after the patient's admission to the hospital while the head was slightly lowered. (Cardiac stimulants are of very little value in these conditions of shock following cranial injuries; whiskey, "hot," has occasionally been used in the enemata and with doubtful value.) The röntgenogram was rather surprising in that a fracture of the squamous portion of both temporal bones was revealed; the bilateral mastoid ecchymoses, however, was of positive value, but only the left tympanic membrane had been lacerated, and it was only in the left ear that the hearing remained slightly impaired—even 27 months following the injury; the fact that it has persisted this length of time would be indicative of its permanent character.

CASE 29.—Acute fracture of base of skull; marked increase of intracranial pressure. No operation. Doubtful recovery.

No. 90.—Edna. Thirty-two years. White. Married. Housewife. U. S.

Admitted September 19, 1914, Muhlenberg Hospital, Plainfield, N. J. Referred by Doctor G. W. Endicott.

Discharged October 14, 1914—25 days after injury.

Family history negative.

Personal History.—Very athletic; was formerly an amateur tennis champion (singles).

Present Illness.—Eighteen hours ago, while returning from a dance late last night in an automobile, patient was thrown out by a collision with another car; was picked up unconscious, and only regained consciousness during the past 6 hours. Has bled copiously from both ears, particularly the left one.

Consultation with Doctor Endicott (18 hours after injury).—Temperature, 99°; pulse, 104; respiration, 24; blood-pressure, 126. Semiconscious and irrational. No paralyses. Still bleeding profusely from both ears, especially from left ear, and it is chiefly straw-colored serum; double mastoid ecchymoses. When aroused by supra-orbital pressure, patient complains of intense headache. Pupils equal and react normally. Reflexes not exaggerated and equal; tendency to a right Babinski; abdominal reflexes—right less than left. Fundi—retinal veins dilated; no definite blurring of nasal margins or halves of the optic disks. Lumbar puncture—bloody cerebrospinal fluid spurted about 8 inches under high tension (no measurement of pressure taken). Almost 40 c.c. of the cerebrospinal fluid carefully removed in order to lessen the increased intracranial pressure and thus to hasten the cessation of the aural discharge and thereby lessen the danger of aural infection; upon standing over night, this cerebrospinal fluid was one-fifth blood.

Treatment.—Expectant palliative. (From the hospital reports, the patient made an excellent recovery—being discharged 25 days after admission.)

Examination (December 10, 1914—3 months after injury).—Complains of headache and dizziness; impairment of hearing—left more than right. Pupils equal and react normally. Reflexes exaggerated but equal; no Babinski. Fundi—retinal veins enlarged; distinct blurring of the nasal margins of both optic disks. Otoscopic examination reveals an irregular scar upon the posterior portion of each ear drum; bone conduction is greater than air conduction, especially in left ear.

Treatment.—General hygiene; patient advised to live quietly and keep out of all active life.

Examination (October 21, 1917—37 months after injury).—Patient has never been really well since the injury; frequent headaches; unable to "play" tennis or other vigorous games for more than one-half hour on account of dull headache and dizzy spells; patient, however, has never fainted. Has become very nervous—unable to sleep and has periods of marked depression. Pupils equal and react normally. Reflexes very active but otherwise negative. Fundi—retinal veins slightly enlarged; no distinct obscuration of the optic disk margins.

Last Report (September 2, 1918—60 months after injury).—Practically the same condition as at the last examination continues. Patient's life becoming more and more isolated; she shuns her former friends and wishes to be alone. Frequently complains of an indefinite pain in the head.

Remarks.—At the time of the first examination of this patient—18 hours after the injury—there was such a profuse discharge of bloody cerebrospinal fluid in both ears that it was believed that the patient would "decompress" herself by lessening the increased intracranial pressure by means of this continuous aural discharge; it must have been a large factor in preventing the increased intracranial pressure from becoming extreme and thus necessitating a subtemporal decompression in order to save the life of the patient or to cause a marked ultimate impairment, but it would seem that this means of decompression and drainage through the ears alone had not been entirely satisfactory in this patient, and I now believe that the subtemporal decompression and drainage would probably have obtained a better ultimate recovery of this patient's good health and normality.

The definite danger of infection extending into either middle ear and then through the line of fracture of the adjacent bone intracranially was fortunately avoided; this is the real danger in those patients in whom the aural discharge continues for several days, and as an excellent means to hasten its cessation and also at the same time to lessen (even though temporarily) the increased intracranial pressure, repeated lumbar punctures may be performed daily; it is surprising how quickly the discharge of cerebrospinal fluid from the ear will cease and in this manner the risk of an otitis media and its complications is minimized.

CASE 30.—Acute fracture of base of skull; marked signs of an increased intracranial pressure. No operation. Doubtful recovery.

No. 244.—Arthur. Twenty-four years. White. Single. Clerk. U. S.
Admitted May 10, 1915, Polyclinic Hospital.
Discharged July 20, 1915—40 days after injury,
Family history negative.
Personal history negative.

Present Illness.—While riding on the rear seat of a motor cycle, patient was struck by a passing automobile; immediate loss of consciousness; brought to the hospital in the automobile.

Examination upon admission (20 minutes after injury).—Temperature, 98.4°; pulse, 88; respiration, 26; blood-pressure, 124. Semiconscious and in shock. Profuse hemorrhage from right ear while bloody cerebrospinal fluid welled out of left ear; double mastoid ecchymoses. Multiple lacerations of scalp, particularly about the occiput. Pupils moderately contracted but equal; normal reaction to light. Reflexes negative. Fundi negative. No thorough examination made at this time on account of the shock.

Treatment.—Expectant palliative; entire head shaved, lacerations cleansed and a warm wet bichloride (1–5000) dressing applied.

Examination (18 hours after admission).—Temperature, 100°; pulse, 82; respiration, 24; blood-pressure, 136. Patient stuporous; irrational when aroused; noisy. Discharge from right ear has ceased but left ear still discharging bloody cerebrospinal fluid. Definite weakness of left side of face (peripheral type). Pupils equal and react normally. Reflexes exaggerated equally and not associated with ankle clonus nor Babinski. Fundi—entire retinæ congested and the nasal margins of optic disks obscured; retinal veins enlarged. Lumbar puncture—cerebrospinal fluid blood-tinged and under increased pressure (approximately 12 mm.). X-ray (Doctor A. J. Quimby)—"no line of fracture observed."

Treatment.—Expectant palliative; an operation of subtemporal decompression would possibly have benefited this patient very much indeed, but he made such a quick and marked improvement in the hospital that it was thought advisable not to perform it; this was the time, however, that an operation should have been performed, judging from the subsequent history of the patient.

Examination (5 days after admission).—Temperature, 100°; pulse, 86; respiration, 22; blood-pressure, 136. Patient has been trying to get out of bed, very noisy and irrational at times, but this morning he is more quiet and seems perfectly conscious; complains of pain in the head and inability to see out of the left eye. Straw-colored discharge from left ear; otoscopic examination of right ear reveals a large tear of the tympanic membrane. Left facial weakness of peripheral type persists. Pupils equal and react normally. Reflexes equally exaggerated but otherwise negative. Fundi—blurring along nasal margins of both optic disks; left disk is paler than right (the earliest beginning of a primary optic atrophy due to a direct injury of the optic nerve itself).

Treatment.—Expectant palliative.

Examination (20 days after admission).—Temperature, 101.4°; pulse, 88; respiration, 24; blood-pressure, 134. Feels better in every way; still pain over left side of head. Discharge of greenish yellow pus with an offensive odor from left ear. (Absolute quiet in bed advised with the usual dietary regulation; no irrigation of the ear permitted—a most dangerous procedure in these cases; merely a sterile gauze pad loosely applied over ear.) Reflexes active but otherwise negative. Fundi—blurring of nasal margins persists, right possibly greater than left; left optic disk, however, definitely paler than right optic disk.

Examination at discharge (40 days after admission).—Temperature, 99°; pulse, 80; respiration, 20; blood-pressure, 134. Feels well, although he complains of slight headache, blindness of left eye and inability to smell. Purulent discharge from left ear has ceased; otoscopic examination reveals a laceration along the inferior margin of tympanic membrane which is very much thickened and fibrous. Weakness of left side of the face still persists. Patient unable to smell and associated with its usual concomitant impairment—the inability to taste well. Pupils equal and react normally; occasional nystagmoid twitch to left. Reflexes active but otherwise negative. Fundi—slight edematous blurring along nasal margin of right optic disk; left optic disk pale with distinct and clear margins.

Examination (June 12, 1917—25 months after injury).—Impairment of vision of left eye persists and impairment of smell; also complains of loss of impairment of hearing of left ear; otherwise feels well and is able to work. Left facial weakness has almost entirely disappeared. Reflexes active but not abnormal. Fundi—left optic disk small and pale—primary optic atrophy; right fundus negative. Hearing of left ear impaired; bone conduction greater than air conduction.

Last Examination (March 12, 1918—34 months after injury).—Preceding complaints persist; patient has just been rejected by the draft board on account of impairment of vision of left eye, impairment of hearing, particularly of the left ear, and loss of the sense of smell. Reflexes active but otherwise negative. Fundi—left primary optic atrophy; right fundus negative.

Remarks.—The ultimate condition of this patient might have been hastened and also improved if an early lessening of the intracranial pressure had been secured early by the operation of subtemporal decompression and drainage; at the time, there was such a profuse discharge of blood from the right ear and of bloody cerebrospinal fluid from the left ear, which continued for several days—so much so that the patient was in this way considered as "decompressing" himself through the ears, and it was thought that the means of natural absorption would then be sufficient for whatever increased pressure of cerebral edema was left; this entire lessening of the increased intracranial pressure was finally obtained after a period of months.

The definite and prolonged weakness of the left side of the face and then its gradual and almost ultimate recovery would indicate a severe compression edema of the left facial nerve in the aqueduct of Fallopius due to a fracture of the adjacent bone. It is rare that these basal fractures of the skull actually produce a complete tear of either facial nerve, although it is possible and does occasionally occur; the great majority of patients, however, merely have a temporary edema of the facial nerve itself within its narrow canal—aqueduct of Fallopius.

The left primary optic atrophy is probably due either to a complete tear of the optic nerve itself anterior to the optic chiasm, or to a bony compression of it in the same location and of sufficient compression to produce a primary optic atrophy. If the site of the lesion were posterior to the optic chiasm and in the left optic radiations then a right homonymous hemianopsia of both eyes would have occurred, whereas if the lesion were situated in the sella turcica and sufficient to destroy the optic chiasm, either by a tear or by severe bony compression, then there would have been total blindness of both eyes.

The left purulent otitis media following an infection extending through the laceration of the left tympanic membrane was a source of great danger to the patient for fear that the infection might extend through the line of fracture of the adjacent bones and the possibility of a purulent meningitis and brain abscess formation resulting; fortunately the patient made an excellent recovery.

CASE 31.—Acute fracture of base of skull; marked signs of an increased intracranial pressure. No operation. Doubtful recovery.

No. 535.—Michael. Forty-four years. White. Married. Driver. Ireland.

Admitted March 25, 1916, Polyclinic Hospital.

Discharged May 7, 1916—42 days after injury.

Family history negative.

Personal History.—Patient has been a heavy drinker.

First Injury.—Nine years ago, patient fell upon his head, a distance of 6 feet, into an areaway; immediate loss of consciousness for 3 days; motor aphasia lasting 5 days; apparently no other signs of intracranial lesion; returned to work 2 weeks after injury.

Second Injury.—Eight years ago severe laceration of scalp of top of head, but no loss of consciousness; was able to work the following day.

Third Injury.—Two years ago, patient fell, breaking right tibia; no signs of head injury.

Present Illness.—Patient was found lying unconscious in the street; his horse and wagon were standing about 100 yards from the place of accident; brought to the hospital in the ambulance.

Examination upon admission (approximately 1 hour after injury).—Temperature, 97.6°; pulse, 76; respiration, 20; blood-pressure, 138. Profoundly unconscious. Extensive laceration over the occiput. Bleeding from the mouth due to 3 teeth having been knocked out; no bleeding from nose or ears; right mastoid ecchymosis. Pupils dilated and react to light sluggishly. Reflexes cannot be obtained; no Babinski. Fundi negative. Lumbar puncture—cerebrospinal fluid blood-tinged and under slightly increased pressure (approximately 11 mm.).

Treatment.—Expectant palliative; scalp laceration widely shaved, cleansed and sutured loosely; 2 drains of rubber tissue inserted.

Examination (16 hours after admission).—Temperature, 99.8°; pulse, 70; respiration, 20; blood-pressure, 142. Semiconscious; can be aroused easily and patient yells and attempts to get out of bed. Marked right mastoid ecchymosis; otoscopic examination reveals the right tympanic membrane bluish and bulging; left tympanic membrane of normal appearance. Slight weakness of right side of face (peripheral type). Pupils equal and react normally. Reflexes—knee-jerks exaggerated but equal; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi—retinal veins enlarged; entire retinæ congested but only the nasal margins of both optic disks obscured by edema. Lumbar puncture—cerebrospinal fluid blood-tinged and under a definite increase of intracranial pressure (approximately 15 mm.). X-ray (Doctor W. H. Stewart)—"wide line of fracture of right occipital bone extending just to the right of the midline forward and posterior to the right mastoid area; old periostitis of vertex of skull" (Fig. 61).

Treatment. — Expectant palliative; no operation was advised in the belief that the patient could "take care of" the intracranial condition of increased pressure of hemorrhage and edema by absorption. (I feel now, however, that a decompression and drainage operation at this period would have resulted in a greater ultimate improvement than was obtained by the expectant palliative method.)

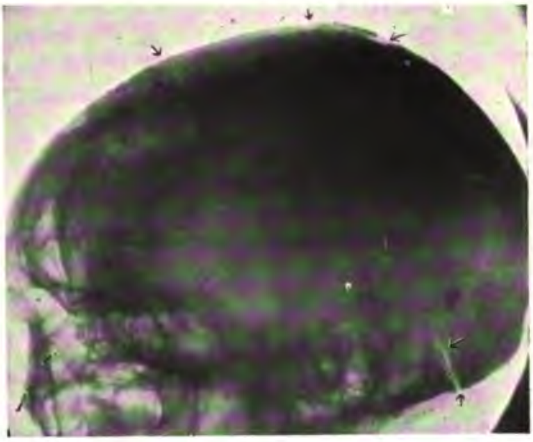

FIG. 61.—Linear fracture of right half of occipital bone; a chronic periostitis of vertex of vault also disclosed. A definite increase of the intracranial pressure treated by the expectant palliative method. Doubtful recovery.

Examination (6 days after admission).—Temperature, 99.4°; pulse, 72; respiration, 18; blood-pressure, 140. Conscious; complains of severe headache "all of the time"; mentally confused at times, noisy and disturbs the other patients. Right mastoid ecchymosis still marked. Right facial weakness has disappeared. Pupils equal and react normally. Reflexes very much exaggerated but otherwise negative. Fundi—dilatation of retinal veins; blurring of nasal margins of optic disks persists. Lumbar puncture—cerebrospinal fluid straw-colored and under an increased pressure (approximately 14 mm.); laboratory report (Doctor Jeffries)—"numerous red blood-cells;" Wassermann test—negative. Expectant palliative treatment continued.

Examination (20 days after admission).—Temperature, 99.4°; pulse, 76; respiration, 18; blood-pressure, 144. Conscious; complains of severe headache; very drowsy and at times slightly irrational. Right mastoid ecchymosis has disappeared; right facial weakness cannot be elicited by special tests. Hearing—right ear impaired. Scalp laceration has healed. Reflexes very active but otherwise negative. Fundi—edematous blurring of nasal margins of optic disks still observed; general congestion of both retinæ, however, has disappeared.

Treatment.—On account of the occasional mental confusion of the patient, he was permitted to remain in the ward for a period of about 8 weeks in the hope that his condition could be thus observed and improved; merely dietetic measures were used in addition to the routine expectant palliative treatment.

Examination at discharge (42 days after admission).—Temperature, 99°; pulse, 76; respiration, 18; blood-pressure, 138. Patient still complains of dull headache, especially when stooping and after eating; speaks very slowly, drawling out the words as in a half-daze; sleeps heavily and for 12 and 14 hours at a time. Hearing of right ear impaired—bone conduction being greater than air conduction. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—slight edematous blurring of nasal margins of both optic disks; retinal veins still enlarged.

Examination (September 20, 1917—18 months after injury).—Patient looks alcoholic and has an alcoholic breath. Unable to work daily, as some days "my head is bad." Complains of headaches, dizzy spells and loss of memory. Wife states that patient becomes drunk at least once a week—his suit for damages having been settled out of court. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—general retinal congestion, but no marked blurring of margins of optic disks. Urine examination—trace of albumen but no casts found.

Last Report (September 10, 1918—30 months after injury).—Wife states that the patient was taken to alcoholic ward in Bellevue Hospital one week ago and that he has just been transferred to Blackwell's Island for a period of 3 months; he had been drinking heavily during the past summer, threatening his wife with bodily injury during quarrels so that she was obliged to have him committed. She now, however, wishes me to help her get him out, but I advised her to let him remain and recommit him if necessary.

Remarks.—This patient should undoubtedly have received the benefit of an early subtemporal decompression and drainage, and particularly following the examination, 16 hours after admission when the increased intracranial pressure was approximately 15 mm.; this was a particularly interesting observation in that at the preceding examination upon admission to the hospital and while the patient was in a mild condition of shock, the intracranial pressure was only slightly increased (approximately 11 mm.), and the fundi were negative, whereas at this examination (16 hours later) not only were there now signs of pressure in the fundi as revealed by the ophthalmoscope, but the measurement of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer had risen to approximately 15 mm. It was believed at this time that the expectant palliative treatment would be sufficient in aiding the absorption of this increased pressure of free hemorrhage and cerebral edema by natural means, and this belief was strengthened by the measurement of the intracranial pressure five days later (6 days after admission), when it was found to be lowered to approximately 14 mm.; unfortunately a later measurement was not taken because the patient seemed to be improving "as well as could be expected"—a very indefinite and vague opinion.

The appearance of the right facial weakness of the peripheral type (the involvement of the right forehead muscles appearing gradually after 16 hours following the injury) would indicate merely an edema of the right facial nerve, due to the proximity of the line of fracture of the petrous bone, or at least a lesion of the neighboring structures; the otoscopic examinations revealing blood in the right middle ear are very suggestive of a fracture of the adjacent bone, and this in itself would be sufficient to produce an edematous compression of the right facial nerve within its narrow bony canal of the aqueduct of Fallopius. The disappearance of this facial weakness within 6 days following the injury confirms the opinion that it was due merely to a temporary edematous constriction of it.

The X-ray report of a fracture of the right occipital bone, and particularly subtentorial, is very impressive in that these subtentorial fractures are most dangerous ones, as they cause so frequently subtentorial hemorrhage and particularly medullary compression and edema when they radiate down to the foramen magnum. It is fortunate for these patients to survive a period of 12 hours following the injury.

Case 32.—Acute fracture of base of skull; marked signs of an increased intracranial pressure. No operation; repeated lumbar punctures. Doubtful recovery.

No. 554.—Joseph. Forty-one years. White. Married. Laborer. Italy. Admitted March 28, 1916, Polyclinic Hospital.

Discharged April 16, 1916—18 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While patient was working upon a scaffold, he fell from a height of 25–30 feet to the ground; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (35 minutes after injury).—Temperature, 98.8°; pulse, 74; respiration, 18; blood-pressure, 138. Well-developed and nourished. Unconscious. Bleeding profusely from left ear; left mastoid ecchymosis. Pupils dilated equally. Reflexes could not be elicited—all absent; cerebrospinal fluid blood-tinged and under high pressure (approximately 15 mm.). (It is rare for this degree of pressure to occur within 6 hours after the head injury—due to the presence of initial shock associated with head injuries—that is, the blood-pressure being low from the shock, naturally it would be difficult for intracranial bleeding to occur unless the torn vessel was an unusually large one; in this patient the blood-pressure was 138 and therefore it was possible for intracranial bleeding to occur early.)

Treatment.—Expectant palliative.

Examination (14 hours after admission).—Temperature, 100.2°; pulse, 70; respiration, 18; blood-pressure, 136. Semiconscious and stuporous. Bleeding from left ear has ceased; otoscopic examination reveals a tear of the posterior portion of left tympanic membrane; right tympanic membrane bluish and bulging, therefore, blood in the right middle ear. Ecchymosis of both mastoid areas—right more than left. Pupils equal and react normally. Reflexes—knee-jerks exaggerated equally; no ankle clonus nor Babinski; abdominal reflexes depressed equally. Fundi—retinal veins dilated; edematous blurring of nasal margins of both optic disks. X-ray (Doctor J. A. Quimby)—"no fracture can be observed."

Treatment.—Expectant palliative; another lumbar puncture and removal of 18 c.c. of blood-tinged cerebrospinal fluid performed in the hope that sufficient drainage and lessening of the intracranial pressure could be thus obtained. These lumbar punctures were repeated daily upon five consecutive days with much relief to the patient—especially a lessening of the headache, the stupor and mental confusion.

Examination (6 days after admission).—Temperature, 99.8; pulse, 72; respiration, 18; blood-pressure, 140. Conscious; complains of a severe frontal headache. Hearing—right ear more impaired, bone conduction being greater than air conduction. Pupils equal and react normally. Reflexes exaggerated but equal—otherwise negative. Fundi—nasal margins of both optic disks blurred; retinal veins enlarged.

Treatment.—Expectant palliative continued.

Examination at discharge (18 days after admission).—Temperature, 98.8°; pulse, 78; respiration, 20; blood-pressure, 142. Complains of headache and some nausea after eating; spells of lightheadedness. Hearing of both ears impaired—right possibly greater than left (due to a blockage of blood in the middle ear—the right tympanic membrane not having been punctured); bone conduction greater than air conduction. Reflexes active but otherwise negative. Fundi—nasal margins indistinct and blurred; retinal veins enlarged.

Examination (October 20, 1917—19 months after injury).—Patient able to work as night watchman—his duties being light. Complains of headache, inability to sleep and easily tired upon the least exertion. Hearing—right ear less acute than left; bone conduction equals air conduction in left ear. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—retinal veins enlarged; a slight edematous blurring along the nasal margins of both optic disks—not so distinct as upon discharge.

Last Examination (July 10, 1918—27 months after injury).—Still working as night watchman and is apparently satisfied with his position. "Pain in the head sometimes but not very bad." Is sleeping better; unable to do any heavy work. Hearing—left ear almost normal; air conduction greater than bone conduction, whereas in right ear bone conduction is still greater than air conduction. Reflexes active but otherwise negative. Fundi—obscuration of nasal margins still persists; practically no enlargement of retinal vessels.

Remarks.—The fact that the signs of an increased intracranial pressure, most probably due to a chronic cerebral edema, have persisted until the last examination would indicate that a subtemporal decompression with drainage would have been the better method of treatment for this patient, and since this increased pressure has persisted this length of time, the hope of its being eventually absorbed by natural means is very remote; in the meantime, as the result of this increased intracranial pressure, the patient is being impaired—a mental retardation and an emotional instability; the sense of early fatigue and the general physical weakness are characteristic of all these patients.

The signs of initial shock so common in these patients having cranial injuries were overshadowed and submerged by the signs of increased intracranial pressure; the shock, however, could not have been severe because the blood-pressure was 138, which would have been impossible in the state of severe shock as the resulting lowered blood-pressure of shock could not have produced a marked increase of the intracranial pressure even if a large intracranial vessel had been torn; it is only when the shock is mild or is disappearing that it is possible for a marked increase of the intracranial pressure to occur.

Although the X-ray was negative for fracture of the skull (and it is rarely possible for a fracture of the base, and particularly of the middle fossa, to be shown by the X-ray), yet the fact that the left tympanic membrane was ruptured and associated with a left mastoid ecchymosis, and that there was a hemorrhage in the right tympanic cavity as revealed by the bluish discoloration of the right tympanic membrane at the otoscopic examination and also associated with a right mastoid ecchymosis—these observations would indicate a fracture of the base of the skull extending through the middle fossa, or at least in the petrous bones.

The impairment of hearing, and especially the observation that the hearing of the right ear was more impaired than that of the left, and the fact that it was the right ear which had the hemorrhage in the tympanic cavity and yet the right tympanic membrane was intact but bluish and bulging—this observation is interesting in that there must have been a greater blockage to the sound transmission mechanism of the right middle ear than of the left middle ear, even though in the latter case the left tympanic membrane had been lacerated. The persistence of a greater impairment of hearing of the right ear would tend to indicate that it would be better judgment to perform a paracentesis of the tympanic membrane in patients where a hemorrhage into the middle ear has occurred and yet the tympanic membrane remains intact; the danger, however, of a possible infective process reaching the middle ear through an incised tympanic membrane and thus extending through the line of fracture to the meninges and even beyond—this complication should be carefully considered before attempting to secure the more acute hearing; ordinarily, however, the danger of infection is small and it should not occur if the proper precautions of asepsis and sterilization are employed.

The marked temporary improvement following each lumbar puncture and withdrawal of 15–20 c.c. of cerebrospinal fluid possibly made us more hopeful of the ultimate good result to be expected in this patient than we should have been; if a subtemporal decompression and drainage had been performed at the time the second lumbar puncture was advised as a therapeutic measure, this patient would now, I believe, be a well man. From this standpoint, the advocacy of repeated lumbar punctures as a therapeutic measure should be limited to the mild cases of increased intracranial pressure, and it must not be considered as a substitute for the cranial operation of subtemporal decompression and drainage.

CASE 33.—Acute fracture of base of skull; signs of a marked increase of intracranial pressure. No operation; repeated lumbar punctures. Doubtful recovery.

No. 581.—Daniel. Thirty-three years. White. Single. Advertising. United States.

Admitted May 16, 1916, Polyclinic Hospital.

Discharged May 28, 1916—12 days after injury.

Family history negative.

Personal history negative.

Present Illness.—Patient was found lying in the street at 2.30 A.M. in a condition apparently of acute alcoholism; unconscious; brought to the hospital in the ambulance.

Examination upon admission (1 hour after injury).—Temperature, 97.6°; pulse, 72; respiration, 18; blood-pressure, 128. Unconscious and in shock. Stertorous, heavy respiration; alcoholic breath. Extensive laceration over left parietal area and a stab wound of right arm above the elbow. Bleeding from right ear, nose and mouth. Pupils very small, but equal; do not react to light. Reflexes—knee-jerks diminished but equal; no ankle clonus nor Babinski; abdominal reflexes absent. Fundi negative except for a general retinal suffusion and congestion.

Treatment.—Expectant palliative; shock treatment instituted.

Examination (16 hours after admission).—Temperature, 100.2°; pulse, 84; respiration, 22; blood-pressure, 138. Conscious but very stuporous. Straw-colored fluid oozing from right ear; right mastoid area ecchymosed and boggy. Pupils—both pin-point (no morphia has been administered). Reflexes—knee-jerks active but equal; suggestive Babinski and Gordon reflexes but no ankle clonus; abdominal reflexes present and equal. Fundi cannot be accurately observed on account of the small pupils. Lumbar puncture—bloody cerebrospinal fluid and under a pressure of 11 mm. X-ray (Doctor W. H. Stewart)—"fracture of right squamous bone extending down into right petrous bone."

Treatment.—Expectant palliative continued. (The operation of subtemporal decompression and drainage should have been performed at this time.)

Examination (36 hours after admission).—Temperature, 100°; pulse, 78; respiration, 18; blood-pressure, 140. Conscious; complaining of severe headache—"splitting." Discharge of cerebrospinal fluid from right ear has ceased; otoscopic examination reveals a small tear in the lower posterior quadrant of the right tympanic membrane; left ear-drum of normal appearance. Hearing of right ear definitely impaired—cannot hear watch. Slight weakness of right side of face—peripheral type. Pupils not so small; equal, and react well. Reflexes—left knee-jerks possibly more active than right; still suggestive bilateral Babinski; no ankle clonus; abdominal reflexes—present and equal. Fundi—retinal vessels dilated; nasal halves of the optic disks blurred—right more than left. Lumbar puncture—cerebrospinal fluid bloody and under an increased pressure of approximately 14 mm.

Treatment.—Expectant palliative. A decompression operation should certainly have been advised at this time, but unfortunately it was not considered necessary in the belief that repeated lumbar punctures and drainage of 15–20 c.c. of cerebrospinal fluid each time would be sufficient. This was performed daily upon 4 consecutive days; each time the cerebrospinal fluid was bloody and under a pressure of approximately 12–14 mm.; patient felt better after each lumbar puncture—particularly a lessening of the headache.

Examination at discharge (12 days after admission).—Temperature, 99.4°; pulse, 80; respiration, 18; blood-pressure, 142. Patient very irritable, restless and complains of "shooting headache." He disobeys orders, will not remain in bed, and insists upon going home, because "you are starving me and I can't sleep here." Patient is discharged at his own risk (A. O. R.). Right mastoid area still slightly ecchymosed and tender upon palpation. Weakness of right side of face not elicited by special tests. Pupils rather small but equal, and react well. Reflexes very active but equal; no Babinski nor ankle clonus; abdominal reflexes present and equal. Fundi—retinal vessels still enlarged; edematous blurring of the nasal margins of both optic disks persists. Patient refused another lumbar puncture at this time and said "I won't be cut up."

Treatment.—Relatives advised to keep patient home in bed quietly and on soft diet; daily catharsis and *no alcohol.*

Examination (September 10, 1916—4 months after injury).—Patient has tried to work but had to "give up" each time. "Too much pain in my head and dizzy spells." "Just dead at night." Pupils equal and react normally. Reflexes active but equal; no ankle clonus nor Babinski. Fundi—general congestion of entire retinæ; distinct obscuration of the nasal margins of both optic disks. Patient refused a lumbar puncture.

Examination (October 20, 1917—17 months after injury).—Patient complains of headache and "general tiredness"; is a night watchman "where I can sit and sometimes sleep." Pupils negative. Reflexes active but otherwise negative. Fundi—retinal veins still large; the nasal margins of both optic disks obscured by edema.

Last Examination (August 12, 1918—27 months after injury).—Still complains of headache—particularly in the morning. "Never really well." Brother says he has become a "bum." Pupils negative. Reflexes active but otherwise negative. Fundi—blurring of the nasal margins still persists. Patient advised again to enter hospital for observation, but he has not consented to do so.

Remarks.—The end-result of this patient will apparently be most discouraging—becoming more and more unstable emotionally, definitely impaired mentally, and it will not be surprising if institutional care will eventually be advisable. An early subtemporal decompression and drainage, unilateral, and if necessary, bilateral, should have been performed, but the relatives were so influenced by the patient himself, who was not in a normal mental condition at the time following the injury, so that the operative permission requested could not be obtained.

The delayed weakness of the right side of the face which was not ascertained until 36 hours after the injury would point merely to an edema of the facial nerve itself within the aqueduct of Fallopius; it was only of temporary duration in that no signs of its presence could be elicited at the time of the patient's discharge, ten days later.

The pupillary contraction, which persisted for more than 36 hours following the injury, was undoubtedly due to a bilateral cortical irritation—most probably a subarachnoid hemorrhage and not of sufficient size and compression to produce the paralytic enlargement of the pupils; in some patients, the initial shock is so great that the pupils are dilated even in the presence of a great increase of the cortical irritability, but as soon as the shock lessens then the pupils become contracted, and if the cortical irritation becomes greater and greater until a definite supracortical clot and compression occur, then the pupils gradually enlarge to the point of dilatation and no longer react to light unless sluggishly; a definite inequality in the size of the pupils—such as a very much contracted pupil of ipsolateral cortical irritation or a dilated pupil of ipsolateral cortical compression, can be of great value in the localization of the greater lesion of either hemisphere.

CASE 34.—Acute fracture of base of skull; signs of a marked increase of intracranial pressure. No operation. Doubtful recovery.

No. 814.—Vincent. Twenty-four years. White. Single. Clerk. Canada.
Admitted March 22, 1917, Polyclinic Hospital.
Discharged April 25, 1917—32 days after injury.
Family history negative.
Personal history negative.

Present Illness.—Patient tripped over a rug, falling headforemost into fireplace and striking the back of his head against an andiron; immediate loss of consciousness; brought to hospital in ambulance.

Examination upon admission (80 minutes after injury).—Temperature, 97°; pulse, 60; respiration, 16; blood-pressure, 128. Unconscious; cold, clammy skin and temperature of 97, indicating severe shock (in the presence of a subtentorial injury affecting the medulla and thus retarding the pulse- and respiration-rates). Laceration of two inches long over left occipital area. Profuse hemorrhage and discharge of cerebrospinal fluid from left ear; left mastoid ecchymosis and bogginess. Pupils slightly dilated but equal; reaction to light sluggish. Reflexes—knee-jerks equally exaggerated; double ankle clonus and double Babinski; abdominal reflexes absent. Fundi—retinal veins dilated; optic disks clear.

Treatment.—Expectant palliative; vigorous treatment of shock instituted and careful observation.

Examination (10 hours after admission).—Temperature, 98.8°; pulse, 64; respiration, 20; blood-pressure, 132. Semiconscious; exceedingly restless, requiring morphia and restraint. Discharge of blood and cerebrospinal fluid from left ear not so profuse. Pupils equal and react more actively. Reflexes—knee-jerks exaggerated; bilateral ankle clonus and Babinski persist; abdominal reflexes, however, can be obtained with difficulty. Fundi—retinal veins dilated; distinct edematous blurring along the lower nasal quadrants of both optic disks. Lumbar puncture—cerebrospinal fluid bloody and under increased pressure (12 mm.). X-ray (Doctor G. W. Welton)—"unusually distinct lamboidal suture, and particularly on the left side, as though a diastasis of it had occurred."

Treatment.—Expectant palliative continued. (If an operation to relieve this increased pressure and to drain the intracranial hemorrhage were to be advised, this was the time it should have been performed. It was thought, however, that the intracranial pressure was not sufficiently high to advise an operation in the belief that the normal absorption would "take care of" this condition; the subsequent history, however, makes me feel that an operation at this time would have been the proper procedure—at least it would have given him a chance for a greater ultimate improvement.)

Examination (36 hours after injury).—Temperature, 99.8°; pulse, 66; respiration, 20; blood-pressure, 134. Conscious, but confused mentally; wants to go home—requiring restraint and morphia. Bleeding from left ear has ceased; otoscopic examination reveals an extensive laceration of the entire posterior attachment of left tympanic membrane to the bone; a dark currant-jelly clot is observed extruding through the tear; left mastoid area very much swollen, ecchymotic and extremely tender. Pupils equal and react normally. Reflexes—both knee-jerks exaggerated; exhaustible ankle clonus and only a suggestive bilateral Babinski; abdominal reflexes present and equal. Fundi—nasal half of left optic disk obscured, but only nasal margin of right optic disk blurred; retinal veins engorged and tortuous.

Treatment.—Expectant palliative continued.

Examination (6 days after admission).—Temperature, 99.4°; pulse, 68; respiration, 20; blood-pressure, 132. Perfectly conscious; complains of severe headache, more on left side. Laceration of scalp healing *per primam.* Reflexes very much exaggerated but neither ankle clonus nor Babinski can be elicited. Fundi—nasal margins of both optic disks obliterated by edema; retinal veins dilated and tortuous.

Examination at discharge (32 days after admission).—Temperature, 98.8°; pulse, 66; respiration, 20; blood-pressure, 138. Conscious; slight general headache each day but not severe; "sort of weak all over." Laceration of scalp healed. Hearing of left ear impaired—bone conduction being greater than air conduction and therefore left middle ear involvement. Pupils equal and react normally. Reflexes very active but otherwise negative. Fundi—retinal veins still enlarged; nasal margins of both optic disks indistinct—left more than right.

Report (by letter, May 15, 1917, 2 months after injury).—"Doing well except daily headaches, very irritable and wants to sleep most of the time; is not interested in things as before the injury."

Report (by letter, September 21, 1917, 6 months after injury).—"My condition is a little improved, but far from being myself; headache almost all the time and so light-headed; I have lived up to your diet and instructions."

Last report (by letter, August 12, 1918, 17 months after injury).—"Vincent has never been himself since the accident. Before, he was a hard-working, cheerful boy; now, he complains of his head every day, grumbles at everything, very irritable, and must lie down after any exertion on account of 'pain in my head.' He cannot work and I do not know what to do with him. Would an operation help him now?"

Remarks.—The above question of the mother is a very pertinent one, and yet it is doubtful whether an operation at this time would improve his condition. Naturally, a thorough examination would be necessary to ascertain the presence or not of an increased intracranial pressure; if present, then a decompression would give him a definite chance of being improved—particularly the headache relieved and his general condition so improved that it would be possible for him to return to his work. On the other hand, if no increased intracranial pressure was present, then no operation would be indicated and there would be nothing to be done except the expectant palliative treatment that he is now receiving. These are the patients for whom an operation at the time of the injury might have effected a greater ultimate recovery.

This patient made such an excellent immediate recovery as far as life and his general condition were concerned that, upon the hospital chart, he could have been designated as "Well"; and yet upon following his history for a period of almost one year and a half, we find that he is not well. and it is very doubtful if he will ever enjoy his former good health. Considering the ultimate recovery of these patients, and chiefly the emotional and mental status, it is then that we are impressed by the advisability and the necessity of an early and efficient relief of the increased intracranial pressure as soon as the signs of shock have subsided. It is taking too great a "chance" to permit these patients to absorb the cerebral edema, unless the signs of intracranial pressure are mild, and only these "mild" cases should be treated by the expectant palliative method.

Recent Mild Brain Injuries Associated with a Depressed Fracture of the Vault, with and without a Fracture of the Base of the Skull

A cranial injury of sufficient force to produce a fracture of the vault of the skull so that the line of fracture radiates from the area of contact, usually causes the line or lines of fracture to extend to the base of the skull—the weaker portions, and particularly the middle fossa; in this manner, extensive linear and also depressed fractures of the vault are frequently associated with a fracture of the base of the skull.

All depressed fractures of the vault (with almost no exceptions) should be elevated or removed—and as early as possible after the initial shock of the injury has subsided; if, however, there are present definite signs of a marked increase of the intracranial pressure due either to hemorrhage or cerebral edema and in the presence or absence of a fracture of the base of the skull, then it is better surgical judgment and of much less risk to the patient to perform first, a subtemporal decompression on the same side of the head as the depressed area of bone, and then, at the same operation, to elevate or remove the depressed area of bone. If this method is not followed in patients having a high intracranial pressure, then the elevation and removal of the bony depression of the vault is attended with much danger of damage to the underlying cerebral cortex—and especially if the dura has been torn; the cerebral tissue may be protruded and even extruded into the bony opening and thus a permanent impairment, anatomically and clinically, will result from the damage to this more highly developed portion of the cerebral cortex; if adjacent to the motor areas, then the impairment of paralysis will not be easily overlooked. The history of many of these patients who have undergone this incomplete and improper method of surgical relief is that their condition is worse after the operation than before it—and it has been a correct observation; so much so, that it has been advocated that no depressed fracture of the vault should be elevated or removed unless definite signs of impairment to the underlying cerebral cortex should appear later—and then the operation at that late date. This latter method of expectancy would be the safer treatment to follow in many patients having a high intracranial pressure as compared with the method of attempting only to elevate or remove the depressed area of bone which is frequently associated with much damage to the underlying cerebral tissues, but the most rational and successful method of treatment for these patients is the relief first of the general increase of the intracranial pressure by means of a subtemporal decompression and then the elevation or removal of the depressed area of the vault; in this manner, the general intracranial pressure is lowered over a comparatively silent area of the cerebral cortex, the dura opened and permitted to remain open, and the operative incision can be firmly closed by the temporal muscle so that there is no danger of unsightly and serious complications of cerebral herniæ and fungi.

Naturally, in patients having a depressed fracture of the vault with and without a fracture of the base and *not* associated with a marked increased intracranial pressure—these patients require only the elevation or removal of the depressed area of bone with and without the opening of the underlying dura, according to the pathology there ascertained; also, in the doubtful cases of high intracranial pressure before operation, when the underlying dura at the site of bony depression is found to be exceedingly tense and bulging and the intracranial pressure is therefore much higher than was ascertained before the operation, then it is better surgical judgment not to open the dura, but to perform an ipsolateral subtemporal decompression immediately, and then, if advisable on account of an underlying local lesion, the dura beneath the depressed area of bone may now be safely opened.

If this method of conservative surgical treatment of these patients having depressed areas of the vault is not used, then not only is the immediate danger present of making the condition of the patient worse than before the operation, but the remote effects upon the underlying cerebral cortex adjacent to the former bony depression are very probable in the formation of adhesions in the presence of an unrelieved increased intracranial pressure, and thus the great danger of future cortical irritation producing headache, epileptiform seizures, changes of personality, and even a mental derangement itself. Merely to elevate or remove a depressed area of the vault in the presence of a high intracranial pressure is not only meddlesome surgery but a very dangerous procedure, both in the immediate effects and in the remote results.

Recent fractures of the base of skull associated with depressed fractures of the vault. Two operations.

A. *Intracranial pressure mild; therefore removal of depressed area of bone first, and then a subtemporal decompression was considered advisable. Excellent recovery.*

Case 35.—Acute fracture of base associated with a depressed fracture of the vault of the skull; mild signs of an increased intracranial pressure. Two operations—removal of depressed area of bone first, then a subtemporal decompression. Excellent recovery.

No. 057.—August. Thirty-nine years. White. Married. Carpenter. Austria.

Admitted April 21, 1913, Polyclinic Hospital. Referred by Doctor J. A. Bodine.

Operations April 21, 1913—8 hours after injury. First, removal of depressed area of vault; second, left subtemporal decompression.

Discharged May 6, 1913—15 days after injury and operations.

Family history negative.

Personal history negative.

Present Illness—While working in a new building, patient was struck upon the top of head by a large iron door; loss of consciousness for several minutes; brought to the hospital in the ambulance.

Examination upon admission (35 minutes after injury).—Temperature, 99.4°; pulse, 64; respiration, 18; blood-pressure, 160. Strong muscular man. Semiconscious; replies to questions drowsily and irrelevantly in German. Definite depression of anterior portion of left parietal bone. Bleeding and discharge of cerebrospinal fluid from left ear; definite left mastoid ecchymosis; left orbital ecchymosis and left subconjunctival hemorrhage. Pupils—left pupil larger than right; reaction to light sluggish. Reflexes—knee-jerks active, right greater than left; no ankle clonus, but right Babinski; right abdominal reflexes less active than left. Fundi—retinal veins enlarged; distinct haziness and blurring edema of the nasal halves of both optic disks. Lumbar puncture—cerebrospinal fluid blood-tinged at first, then clear and under an increased pressure (approximately 14 mm.).

Treatment.—No shock being present and the definite signs of a local cerebral impairment and an increase of the intracranial pressure being ascertained, the operative removal of the depressed area of bone advised as early as possible; (at the time, no further operation was considered necessary nor advisable).

Operations (8 hours after admission).—1st. Removal of depressed area of bone: curvilinear incision of 2½ inches over anterior portion of left parietal bone; small trephine opening made at posterior and upper margin of depressed area; opening enlarged by rongeurs to a diameter of 1½ inches. Dura bluish and under high tension; it was not considered advisable to open dura through this small bony opening for fear that the increased intracranial pressure would damage the underlying cortex, so that a homolateral (left) subtemporal decompression was thought necessary.

2nd. Left subtemporal decompression: usual incision, bone removed and no complications. Dura tense and bluish; upon incising it, much blood-tinged cerebrospinal fluid escaped under pressure; numerous small blood-clots—size of ten-cent pieces, welled out of opening and apparently coming from base of the skull. Cortex congested but otherwise negative; pulsation normal at close of operation. Usual closure with 2 drains of rubber tissue inserted. Duration of operations, 75 minutes.

Post-operative Notes.—Uneventful operative recovery; general condition improved immediately.

Examination at discharge (15 days after admission).—Temperature, 98.6°; pulse, 74; respiration, 22; blood-pressure, 152. No complaints, except for soreness over left side of head. Both operative incisions have healed perfectly; decompression site bulges slightly, pulsates normally. Hearing of left ear impaired; bone conduction greater than air conduction. Reflexes very active but otherwise negative. Fundi—retinal veins slightly enlarged; indistinct blurring of lower nasal quadrants of both optic disks; physiological cup shallow from edema and possibly new tissue formation. X-ray demonstrates the area of bony defect of the left vault due to the two operations (Fig. 62).

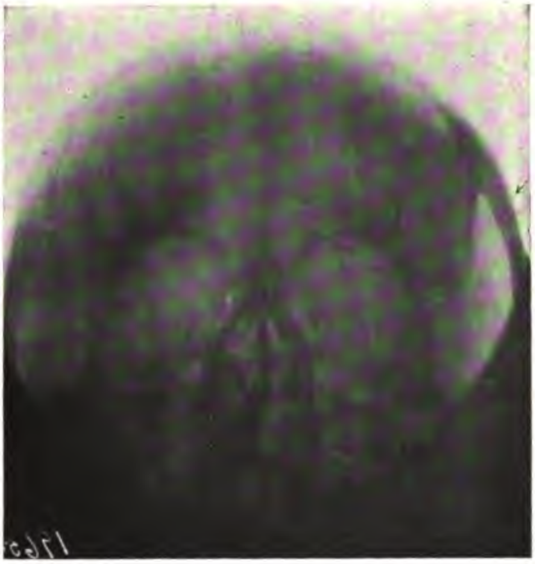

FIG. 62.—Extensive bony defect of left parieto-squamous area of the vault following an operative removal of the bone and a lowering of the increased intracranial pressure. Excellent recovery.

Examination (September 21, 1915—29 months after injury).—No complaints; works daily. Hearing of left ear less acute than right; bone conduction equals air conduction. Reflexes negative. Fundi negative.

Last Examination (May 10, 1918—61 months after injury).—Examination was made at Fort Jay, New York, as patient had just been interned as an enemy alien. No complaints; both operative sites depressed and neither pulsates (due most probably to new bone formation). Hearing of left ear equals acuity of hearing of right ear; tuning fork tests not made, unfortunately. Reflexes negative. Fundi negative.

Remarks.—The operative treatment of this type of patient is now just the reverse; that is, patients having depressed fractures of the vault, with or without a fracture of the base of the skull, and there are definite signs of an increased intracranial pressure, then it is always better surgical judgment to perform the subtemporal decompression first and thus relieve whatever increased intracranial pressure there is, and then at the same operation, remove the depressed area of the vault or at least elevate it; in this manner, the danger of operative damage resulting to the cerebral cortex underlying the depressed fracture of the skull is practically nil and the lessening of the increased intracranial pressure of subdural and subarachnoid hemorrhage, together with the cortical irritation of the depressed area of bone is thus secured. To open the dura underlying the depressed fracture of the vault, and there is a definite increase of the intradural pressure, then there is a great risk of the underlying cerebral cortex being damaged by its protrusion upward into the overlying dural and bony opening and definite clinical symptoms and signs of this cortical impairment being exhibited by the patient later; the danger, too, of convulsive seizures is also increased, especially in the presence of an increased intracranial pressure.

Case 36.—Acute fracture of base associated with depressed fracture of vault of skull; mild signs of an increased intracranial pressure. Two operations—removal of depressed area first, and then a subtemporal decompression. Excellent recovery.

No. 058.—Constantino. Fifty-three years. White. Married. Laborer. Italy.

Admitted April 23, 1913, Polyclinic Hospital. Referred by Doctor J. A. Bodine.

Operations April 23, 1913—4 hours after injury. First, removal of depressed area of bone; second, right subtemporal decompression.

Discharged May 7, 1913—14 days after injury and operations.

Family history negative.

Personal history negative.

Present Illness.—While working in a new building, patient fell a distance of 3 stories, striking upon the top of his head; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (10 minutes after injury).—Temperature, 99.4°; pulse, 84; respiration, 20; blood-pressure, 156. Well-developed and nourished. Patient had regained consciousness in the ambulance and refused to go to bed in the ward—walking about and desiring to go home. Distinct depression of left parietal bone adjacent to the longitudinal sinus. Bleeding and discharge of cerebrospinal fluid from right ear; right mastoid ecchymosis. Pupils—right larger than left and reacts to light sluggishly. Reflexes—knee-jerks exaggerated, left more than right; suggestive left Babinski; left abdominal reflexes possibly depressed. Fundi—retinal veins enlarged—possibly more in right than in left; nasal margins and nasal halves of optic disks obscured by edema. Lumbar puncture—cerebrospinal fluid blood-tinged and under an increased pressure (approximately 13 mm.).

Treatment.—The rapid onset of the marked signs of an increased intracranial pressure (it being rare for definite signs of an increased intracranial pressure to appear within 6 hours after a cranial injury due undoubtedly to the usual association with severe shock and to a less rapid formation of cerebral edema and intracranial hemorrhage) made advisable the immediate removal of the depressed area of bone, and if the intracranial pressure was confirmed to be markedly increased, then a subtemporal decompression and drainage would be indicated—and on the right side on account of the neurological examination indicating a greater impairment of the right cerebral hemisphere.

Operations (4 hours after admission).—1st. Removal of depressed area of vault: through a curvilinear incision of 2 inches overlying the depressed area of the upper portion of left parietal bone (just to the left of the longitudinal sinus), a small trephine opening was made posterior to the depressed fracture; upon removing the small button of bone, a dark currant jelly clot was forced out under very high tension. For fear that the underlying dura had also been torn, it was now thought advisable to perform a right subtemporal decompression to lessen the intracranial pressure and then to remove or elevate the depression. A cotton pad was accordingly placed over the trephine opening to prevent the protrusion of more clot and possibly brain tissue (a most dangerous complication for fear of producing paralysis and other signs of cortical destruction) and a right subtemporal decompression was hurriedly performed.

2nd. Right subtemporal decompression: Usual incision, bone removed and no complications. An extradural currant jelly clot had extended downward to about the middle of the decompression opening; this was removed. Dura tense and slightly bluish; upon incising it blood-tinged cerebrospinal fluid welled out under increased pressure; no distinct subdural clots evacuated. Cortex "wet" and edematous, showing the characteristic arachnoid "sweating." Usual closure with 2 drains of rubber tissue inserted. Attempt was now made to elevate the depressed area of bone, but as the line of fracture extended beyond the longitudinal sinus and as the longitudinal sinus itself was found to be torn and still bleeding profusely, it was considered advisable merely to rongeur away more of the depressed area of bone and then to pack the longitudinal sinus; this was done with 2 small strips of gauze packing which were left *in situ*. Usual closure with 2 drains of rubber tissue inserted. Duration of both operations, 80 minutes.

Post-operative Notes.—Uneventful operative recovery; the gauze strips packing the longitudinal sinus were removed in 48 hours and no bleeding occurred; general condition gradually improved.

Examination at discharge (14 days after admission).—Temperature, 99°; pulse, 76; respiration, 22; blood-pressure, 146. No complaints except for a feeling of fulness, especially over right side of head and impairment of hearing of right ear; otoscopic examination reveals a small tear in the lower posterior portion of the right tympanic membrane—apparently closing; bone conduction greater than air conduction in right ear. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—enlargement of retinal veins persists; a slight blurring of the nasal margins, especially of the right optic disk. X-ray (Doctor A. J. Quimby)—"bony defects due to removal of depressed bone at vertex and to right decompression operation, demonstrated" (Fig. 63).

Examination (October 20, 1915—30 months after injury).—No complaints; returned to work 6 weeks after injury and has been working daily since. Reflexes negative. Fundi negative. Superficial examination reveals no impairment of hearing of right ear.

Last Examination (July 14, 1918—63 months after injury).—No complaints referable to the head injury. Hearing negative; air conduction greater than bone conduction of both ears. Reflexes negative. Fundi negative.

Remarks.—This is another patient upon whom it would have been better surgical judgment to have performed a right subtemporal decompression first, and then the removal or elevation of the vault—and in reality, this order of procedure was used with the exception of the trephine opening at the site of the depression. In the period when this operation was performed (1913), it was not known that the ophthalmoscopic and lumbar puncture examinations could be such accurate indications of increased intracranial pressure as they are now known to be, so that similar patients to this one would now always have the increased intracranial pressure lessened, first by a subtemporal decompression, and then the depressed area of the vault treated accordingly. If this method of procedure is not used, then there are certain patients with high intradural pressure who will be permanently damaged by the operative cortical impairment resulting from the local cortical extrusion at the site of the depressed area of the vault and through the open dura underlying it.

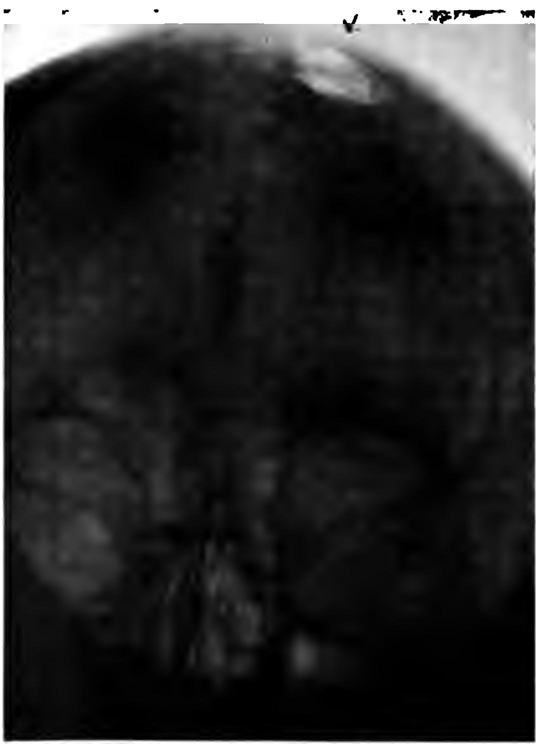

Fig. 6[illegible].—Irregular bony defect of left parietal bone following the removal of bony depression; the increased intracranial pressure lowered by a right subtemporal decompression. Excellent recovery.

It was very interesting in this patient following the operation to observe the rapid subsidence of the signs pointing to a lesion of the right hemisphere: enlarged right pupil resulting from the paralytic effect of compression upon the right cortex, the exaggerated left deep reflexes, suggestive left Babinski and the depressed left abdominal reflexes, while the signs of pressure as exhibited by the retinal veins were greater in the right fundus than in the left; the escape of blood and cerebrospinal fluid from the right ear is of little significance regarding the intracranial condition of the increased pressure which is all-important in these patients. The rapid improvement in the general condition of this patient and the almost immediate disappearance of the symptoms and signs of an increased, intracranial pressure following the operations were most impressive.

Case 37.—Acute fracture of base associated with a depressed fracture of vault; definite signs of a mild increase of the intracranial pressure. Two operations—removal of the depressed area of bone first, and then a subtemporal decompression. Excellent recovery.

No. 145.—Annie. Forty years. White. Married. Housework. U. S. Admitted April 9, 1914, Polyclinic Hospital. Referred by Doctor C. H. Chetwood.

Operations.—April 9, 1914—two hours after injury. First, removal of depressed area of vault; second, left subtemporal decompression.

Discharged April 20, 1914—ten days after injury and operations.

Family history negative.

Personal history negative.

Present Illness.—While at home, patient slipped and fell headlong down a flight of stairs; apparently no loss of consciousness; brought to hospital in the ambulance.

Examination upon admission (one hour after injury).—Temperature, 98.6°; pulse, 90; respiration, 20; blood-pressure, 140. Perfectly conscious and clear mentally; odor of alcohol upon her breath, but not to the degree of intoxication. Extensive laceration of scalp of 3 inches parallel and just to the left of the longitudinal sinus and immediately posterior to the frontal hairline; bleeding profusely and matted with hair; careful probing reveals a depressed fracture of the underlying bone; dark clots and particles of brain tissue protruding through opening. Profuse bleeding from left ear; left mastoid ecchymosis. Pupils equal and react normally. Reflexes: knee-jerks active—right possibly greater than left; no ankle clonus nor Babinski. Fundi negative.

Treatment.—Patient insisted that nothing was the trouble with her and desired to return home; one hour later, in the ward, she complained of being dizzy and having a headache; then became drowsy and stuporous. An immediate removal of the depressed area of bone was considered advisable.

Operations (90 minutes after admission).—First, removal of depressed area of left parietal bone: scalp laceration enlarged at each end downward, thus making a sort of curvilinear flap incision; a depressed fracture just to the left of the superior longitudinal sinus disclosed to be depressed to a depth of one-half inch about which dark blood clots and brain tissue ooze. Small trephine opening made at the lower outer edge of the depression and surrounding depressed bone rongeured away—exposing a large extradural clot the size of a lemon; through the underlying torn dura, small subdural clots extruded. Profuse bleeding occurred due to a tear of the longitudinal sinus which was packed successfully by a small gauze tape packing. Usual closure with 3 drains of rubber tissue inserted. On account of the definite increase of intracranial pressure as shown by the extrusion of brain tissue through the torn dural opening, an immediate homolateral (left) decompression was considered advisable.

Second, left subtemporal decompression: usual incision, bone removed and no complications. Dura tense and bluish; upon incising it almost pure blood spurted a height of 8 inches for 18 seconds; upon enlarging dural opening, subdural clots, the size of silver dimes, welled out of opening. Cortex congested, otherwise apparently normal. Usual closure with 2 drains of rubber tissue inserted. Duration of operation, 80 minutes. Post-operative notes: uneventful operative recovery; general condition promptly improved.

Examination at discharge (10 days after admission).—Temperature, 98.6°; pulse, 78; respiration, 20; blood-pressure, 132. No complaints other than soreness over left side of head. Both operative incisions have healed nicely. Hearing of left ear impaired; otoscopic examination of left ear reveals a large laceration of the entire posterior portion of left tympanic membrane; bone conduction is greater than air conduction in left ear. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—retinal veins slightly enlarged; no definite blurring of the optic disk margins. Decompression area moderately tense. X-ray (Dr. A. J. Quimby)—"bony defects of left subtemporal decompression and removal of depressed area of bone clearly shown" (Fig. 64).

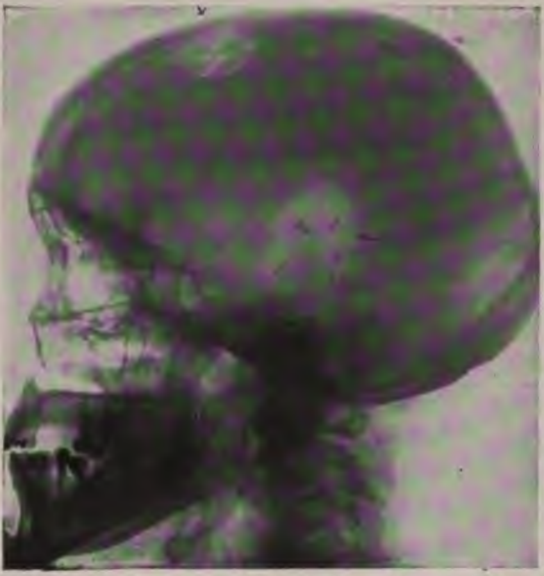

FIG. 64.—Oval bony defect of left subtemporal decompression and irregular bony defect of the removal of the depressed area of left parietal bone demonstrated; an unusual number of silver clips clearly shown in the area of decompression. Excellent recovery.

Examination (September 20, 1916—29 months after injury).—No complaints referable to head injury; patient, however, is becoming alcoholic—similar to her two sisters and one brother. "Never felt better in my life." Hearing of left ear less acute than right; bone conduction equals air conduction. Reflexes difficult to obtain but otherwise negative. Fundi—both retinæ congested but otherwise negative.

Last Examination (June 6, 1918—50 months after injury).—No complaints; patient is no longer drinking, having become a member of a temperance organization and also a suffragette interested in ward politics. "I am now a good Tammany man." Hearing—no impairment elicited. Reflexes sluggish but otherwise negative. Fundi negative.

Remarks.—It is indeed surprising that a person with such a severe cranial condition could walk about in the hospital ward with practically no complaints; it should be remembered, however, that a sort of natural decompression had been performed by the injury itself, so that through the depressed area of the vault the blood clots and even brain tissue could escape, thus relieving the intracranial pressure; to be sure, such a decompression by allowing brain tissue to be extruded is a poor means of lessening the intracranial pressure—the end-result being most disastrous, even if the patient should survive. In all probability, however, the patient would soon have gone into a condition of coma due to the continued hemorrhage and cerebral edema and the rising intracranial pressure resulting from the insufficient drainage, and then the prognosis would have been very grave indeed.

B. *Intracranial pressure very high; therefore, subtemporal decompression first, followed by a removal of depressed area of bone. Excellent recovery.*

Case 38.—Acute fracture of base of skull associated with a depressed fracture of vault; marked signs of an increased intracranial pressure. Two operations—subtemporal decompression first, then removal of depressed area of bone. Excellent recovery.

No. 075.—Martin. Twenty-two years. White. Single. Iron-worker. United States.

Admitted August 11, 1913, Polyclinic Hospital. Referred by Doctor John P. Grant.

Operations (August 11, 1913—5½ hours after injury).—First, right subtemporal decompression; second, removal of depressed area of bone.

Discharged August 24, 1913—13 days after injury and operations.

Family history negative.

Personal history negative.

Present Illness.—While walking along the street, patient was struck upon the head by an iron bar which had fallen from a chimney of 5 stories high; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (30 minutes after injury).—Temperature, 98.8°; pulse, 72; respiration, 22; blood-pressure, 128. Well-developed and nourished youth. Unconscious but not in severe shock. Deep irregular laceration over right frontal bone extending beyond the median line; gentle probing reveals a distinct fracture of the underlying bone. Profuse bleeding and discharge of cerebrospinal fluid from right ear; extensive right mastoid ecchymosis. Pupils equal and react normally. Reflexes—knee-jerks can just be obtained and are equal; tendency to left Babinski; left abdominal reflex absent, whereas right reflex can just be obtained. Fundi—marked dilatation of retinal veins; distinct edematous blurring of nasal halves of both optic disks. (No lumbar puncture performed as the signs of high intracranial pressure were present in both fundi and the discharge of bloody cerebrospinal fluid from the right ear indicated that an intracranial hemorrhage had occurred; at present, a lumbar puncture would be performed to estimate accurately the intracranial pressure by means of the spinal mercurial manometer.) X-ray (Doctor A. J. Quimby)—"small irregular depressed fracture of median portion of frontal bone" (Fig. 65).

Treatment.—As the patient was in excellent condition and did not show the usual signs of shock, an immediate operation was advised to lower this increased intracranial pressure and to remove the depressed bone.

Operations (5 hours after admission).—1st. Right subtemporal decompression (as the intracranial pressure was high it was thought advisable first to perform the decompression, and then to remove the depressed area of bone): usual vertical incision, separation of fibres of the underlying temporal muscle, and removal of bone to a diameter of 2½ inches. Dura very tense and slightly bluish; upon incising it, bloody cerebrospinal fluid spurted to a height of 3 inches. Cortex very congested and edematous—much bloody cerebrospinal fluid escaping so that the brain pulsated normally at the end of the operation. Usual closure with 2 drains of rubber tissue inserted.

2nd. Removal of depressed area of bone: laceration of right frontal area enlarged at each end and retractors inserted exposing a depressed fracture of the right frontal bone extending over the longitudinal sinus and about one-half inch beyond the midline; a small trephine opening made at its lower portion and then the depressed area of bone rongeured upward to the longitudinal sinus, but not beyond, as it was not considered necessary. There was no tear of the underlying dura nor had the longitudinal sinus been torn. Usual closure of scalp with 2 drains of rubber tissue inserted. Duration of both operations, 70 minutes.

Post-operative Notes.—Uneventful operative recovery; immediate general improvement.

Examination at discharge (13 days after admission).—Temperature, 98.8°; pulse, 78; respiration, 22; blood-pressure, 134. No complaints except for soreness over right side of head; general weakness but otherwise "feels fine." Laceration of scalp and decompression incision have healed; site of decompression operation bulges slightly and pulsates. Impairment of hearing of right ear—bone conduction being greater than air conduction; otoscopic examination reveals a small perforation of the posterior portion of right tympanic membrane; a slight right mastoid ecchymosis persists. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—retinal veins enlarged; indistinct blurring along the lower nasal quadrants of both optic disks.

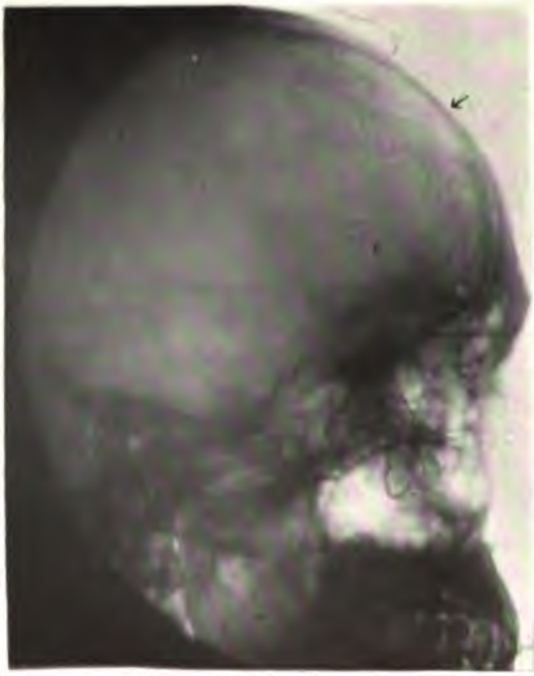

FIG. 65.—Depressed fracture of median portion of frontal bone associated with a high intracranial pressure; therefore a right subtemporal decompression first, to be followed by a removal of the depressed area of the frontal bone. Excellent recovery.

Examination (September 20, 1915—25 months after injury).—Patient has been working as a foreman ever since the accident; "I feel as well as ever." No headache nor dizzy spells; emotionally stable. Hearing of right ear possibly less acute than left; air conduction greater than bone conduction. Reflexes negative. Fundi negative.

Last examination (August 26, 1918—60 months after injury).—Patient came to see me just before embarking for France in the American Expeditionary Force; "the army doctors never noticed my head and I did not tell them." No complaints. Sites of operations depressed and apparently new bone has formed over the median frontal area. Hearing negative. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi negative.

Remarks.—Whenever there are definite signs of an increased intracranial pressure associated with a depressed fracture of the vault, it is always better surgical judgment to perform a homolateral subtemporal decompression first, and then to elevate or remove the depressed area of the vault and thus avoid the complication of operative damage to the underlying cerebral cortex which otherwise might be extruded through the dural opening by the high intradural pressure; in the presence of a marked increase of the intracranial pressure and the dura is not opened but merely the depressed area of bone elevated and removed, the benefits of such a procedure would be very small indeed, if any; the dura must always be opened in order to lessen the increased intracranial pressure—the dura being inelastic in adults.

It was very impressive in this patient following operation to observe the immediate subsidence of the signs pointing to an irritative and compressive lesion of the right hemisphere—the suggestive left Babinski, the absent left abdominal reflex and the immediate cessation of the discharge of blood and cerebrospinal fluid from the right ear. The danger of possible infection through this latter source is always greatly lessened by an early cranial decompression and drainage.

It is very gratifying to ascertain in these patients having a definite impairment of hearing referable to the middle ear and resulting from the fracture of the adjacent bone and the consequent laceration of the tympanic membrane, that this impairment of hearing almost invariably markedly improves and in many patients the hearing returns to normal acuity following a normal natural repair of the laceration of the tympanic membrane; usually within a period of a year, the improvement is a very definite one. It is only in those patients whose auditory nerve has been irreparably damaged, either by complete severance or by bony compression of it and thus the impairment of hearing being referable to the auditory nerve itself, that little or no improvement occurs and the end-result is bad.

CASE 39.—Recent fracture of base associated with compound depressed fracture of vault of skull; definite signs of high intracranial pressure. Two operations—subtemporal decompression first, and then a removal of depressed area of bone. Excellent recovery.

No. 075.—Luigi. Twenty-eight years. White. Married. Laborer. Italy.

Admitted June 30, 1913, Polyclinic Hospital. Referred by Doctor Alexander Lyle.

Operations (June 30, 1913—7½ hours after injury).—1st. Right subtemporal decompression; second, removal of depressed area of bone.

Discharged July 21, 1913—21 days after injury and operations.

Family history negative.

Personal history negative.

Present Illness.—While working in a new building, patient was struck upon the head by a wheelbarrow loaded with bricks falling a distance of 4 stories; immediate loss of consciousness for several minutes; brought to the hospital in a truck.

Examination upon admission (35 minutes after injury).—Temperature, 100°; pulse, 66; respiration, 16 (slightly irregular); blood-pressure, 144. (Another example of retardation of pulse- and respiration-rates in a patient having an occipital injury.) Semiconscious. Laceration of scalp over right occipital bone; gentle probing and retraction of scalp edges exposes a depressed fracture of the underlying occipital bone. Bleeding from right ear; right mastoid ecchymosis. Pupils equal and react normally. Reflexes—knee-jerks exaggerated, especially left; suggestive left Babinski; abdominal reflexes present and equal. Fundi—retinal veins full and slightly tortuous; the nasal halves of both optic disks obscured by edema. (No lumbar puncture performed. No X-ray taken.)

Treatment.—On account of the increased intracranial pressure and the absence of shock, an immediate operative relief of the intracranial pressure with drainage and then the elevation or removal of the depressed area of bone were advised.

Operations (7 hours after admission).—1st. Right subtemporal decompression: usual incision and bone removed; no complications except the bone was very thick and hard. Dura slightly bluish and under a high tension; upon incising it, blood-tinged cerebrospinal fluid spurted to a height of 7 inches. Cortex tended to protrude, being very "wet" and edematous; arachnoid continued to "sweat" throughout the operation, so that much cerebrospinal fluid escaped and thus lessened the cerebral tension. Usual closure with 2 drains of rubber tissue inserted.

Examination (April 21, 1916—34 months after injury).—No complai Reflexes negative. Fundi negative.

Last Examination (August 10, 1918—62 months after injury).—No c plaints. Operative areas depressed and a layer of hard new bone forma is present. Hearing negative; air conduction greater than bone conduc in both ears. Reflexes negative. Fundi negative.

Remarks.—It is unfortunate that no lumbar puncture was made in patient; the importance of estimating accurately the degree of increa intracranial pressure was not then fully appreciated—it was then mo question of ascertaining the presence or not of blood in order to assert greater certainty that a fracture of the skull was present (a belief known to be most fallible and of no great importance in the treatmen the patient—surely not as to the advisability or not of a cranial decomp sion). No X-ray was taken in the belief that a fracture of the skull mus present since there was blood extruding from the right ear throug lacerated right tympanic membrane and associated with a right mas ecchymosis; an X-ray, besides, was not considered necessary because it evident from the probing and palpation that a depressed fracture of vault was present. Whenever possible, however, it is advisable to h cranial röntgenograms made for fear a depression of the vault or ot abnormality might be overlooked.

The unusual high pressure revealed at operation in this patient so t the cerebrospinal fluid spurted to a height of 7 inches is impressive in t frequently the ophthalmoscopic and lumbar puncture tests do not re as high an intracranial pressure as exposed at operation, while they rarel ever err in showing greater intracranial pressure than is really present.

Case 40.—Acute fracture of base associated with a depressed fract of vault of skull; marked signs of an increased intracranial pressure. T operations—subtemporal decompression first, then removal of depres area of bone. Excellent recovery.

No. 081.—David. Sixteen years. White. Single. School. U. S.

Admitted September 11, 1913, Polyclinic Hospital. Referred by Do John A. Wyeth.

Operations (September 12, 1913—16½ hours after injury).—First, ri subtemporal decompression; second, removal of depressed area of bone.

Discharged September 21, 1913—10 days after operations.

Family history negative.

Personal history negative.

Present Illness.—While descending the stairway of the elevated railr patient slipped upon a banana peeling and fell headforemost to the pa ment below—landing upon 2 men; immediate loss of consciousness; brou to the hospital in the ambulance.

Examination upon admission (25 minutes after injury).—Temp ture, 98°; pulse, 72; respiration, 24; blood-pressure, 120. Semiconsci and in shock (though the signs of shock are being overshadowed by the in cranial pressure as indicated by the relatively low pulse-rate). Much vo ing of undigested food. Tender ecchymotic area over right bleeding from nose, mouth or ears; right subconjunctival hemorrhage. Pupils equal and react normally. Reflexes—knee-jerks very sluggish but apparently equal; no ankle clonus nor Babinski; abdominal reflexes not obtained. Fundi—retinal veins enlarged; margins of optic disks clear. Lumbar puncture—cerebrospinal fluid clear and under an increased pressure (approximately 11 mm.).

Treatment.—Expectant palliative; careful observation to ascertain if the signs of high intracranial pressure should appear and thus make it advisable to perform the operation of subtemporal decompression after the signs of shock had subsided.

Examination (16 hours after admission).—Temperature, 99.4°; pulse, 60; respiration, 16; blood-pressure, 136. Conscious; complains of severe headache. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—retinal veins dilated; nasal margins and nasal half of right optic disk obscured by edema. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 14 mm.). X-ray (Doctor A. J. Quimby)—"Y-shaped lines of depressed fracture over right frontal area—extending into right orbit and downward into base" (Fig. 66).

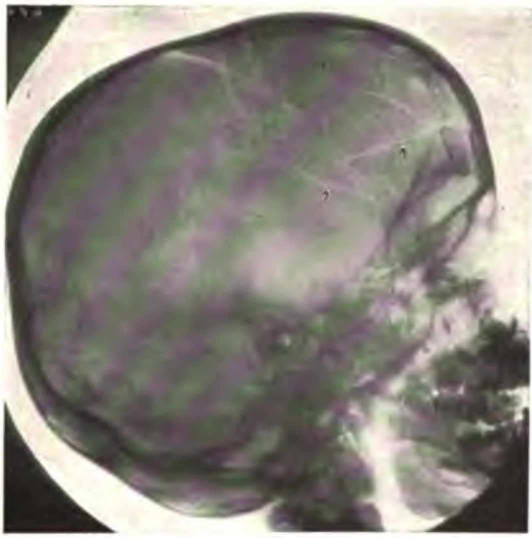

Fig. 66.—Extensive linear and depressed fractures of anterior portion of vault associated with a high intracranial pressure; therefore, a subtemporal decompression first, to be followed by the removal of the depressed area of bone. Excellent recovery.

Treatment.—Owing to the increasing signs of a high intracranial pressure, it was thought advisable to perform a right subtemporal decompression first and then to elevate or remove the depressed area of bone and, whenever possible, through the upper part of the same operative incision.

Operations (18 hours after injury).—1st. Right subtemporal decompression: usual incision, bone removed and no complications; underlying temporal muscle ecchymosed with clotted blood among the muscle fibres (this condition *always* indicates an underlying fracture of the bone and I have yet to see this sign fail; naturally the overlying scalp must not be ecchymotic, otherwise a local injury could produce a similar condition); upon retracting the separated muscle fibres a transverse fracture was exposed, branching forward toward the orbit and downward toward the base; the bone was depressed to a depth of 2 cm. Dura very tense and upon incising it, clear cerebrospinal fluid spurted to a height of 7 inches; arachnoidal "sweating" very profuse, allowing the tense edematous cortex to pulsate normally before the end of the operation. Usual closure with 2 drains of rubber tissue inserted.

2nd. Removal of depressed area of bone: small curvilinear incisio
depression of right posterior frontal area; retractors inserted, revea
depressed fracture. Small rongeurs inserted and an area 1 inch in di
removed. Dura not injured and of normal appearance. Usual closur
2 drains of rubber tissue inserted. Duration of both operations, 60 mi

Post-operative Notes.—Excellent operative recovery; the signs
creased intracranial pressure quickly lessened.

Examination at discharge (10 days after admission).—Temper
99°; pulse, 78; respiration, 24; blood-pressure, 130. Patient in excelle
dition and refuses to remain longer in the hospital; no complaints
soreness over right side of head. Right orbit still slightly ecchymose
right subconjunctival hemorrhage still persists. Pupils equal and rea
mally. Reflexes active but otherwise negative. Fundi—nasal marg
both optic disks still blurred but not the nasal halves; retinal veins enl
Decompression area tense and bulges slightly.

Examination (April 23, 1915—19 months after injury).—No compl
graduates from high-school this year. Decompression area depresse
pulsates slightly. Reflexes negative. Fundi negative.

Last Examination (May 23, 1918—56 months after injury).—P
came for examination and to ascertain whether his former head i
would prevent him from entering the army. No complaints. Exce
depressions at sites of former operations, the physical condition is ne
and I see no reason why he should not be admitted to the service. R
negative. Fundi negative.

Remarks.—This case is a good illustration of the increased intrac
pressure being due, not to an intracranial hemorrhage (and it rarely
to hemorrhage alone), but to an acute cerebral edema—either a tem
blockage of the cerebrospinal fluid or an increased secretion of it, and
probably the former. Although it is rather unusual for the cerebro
fluid intracranially to be perfectly clear in the presence of a fr
of both the vault and the base, yet it does occur, and particularly wh
fracture is limited to the vault alone. From the standpoint of trea
and also prognosis, it is a question of increased intracranial pres
whether it is due to hemorrhage or to cerebral edema matters littl
object of the treatment being to lessen this increased pressure, either
natural means of absorption or by the mechanical means of subten
decompression and drainage.

The clinical syndrome of temperature, pulse, respiration and
pressure in these traumatic cases is very significant and of much va
estimating the general condition of the patient, and in a less accurate m
the presence or not of a definite increase of the intracranial pressure.
admission, the temperature being 98° and the blood-pressure 120
indicate the condition of shock, and it would be expected naturally th
pulse- and respiration-rates would be possibly 100 and 30, respectively
ever, the condition of mild shock being associated with a moderate
of increased intracranial pressure as the signs of shock became less an
the pulse- and respiration-rate was only 72 and 24 and thus indicate
the signs of shock were gradually being overshadowed by the sign
increasing intracranial pressure. This opinion is

tion 16 hours later when the temperature was 99.4 and the blood-pressure 136, whereas the pulse- and respiration-rate was 60 and 16 and thus indicating that the condition of shock had been entirely overcome and that the stage of increased intracranial pressure was now definitely established.

CASE 41.—Acute fracture of base associated with depressed fracture of vault of skull; marked signs of high intracranial pressure. Two operations—subtemporal decompression first, and later a removal of the depressed area of bone. Excellent recovery.

No. 727.—Edward. Twenty-seven years. White. Single. Chauffeur. United States.

Admitted November 17, 1916, Polyclinic Hospital.

Operation (first) November 23, 1916—6 days after injury. Left subtemporal decompression.

Operation (second) November 30, 1916—13 days after injury. Removal of depressed area of bone.

Discharged December 16, 1916—29 days after injury and 16 days after second operation.

Family history negative.

Personal history negative.

Present Illness.—While crossing the street, patient was knocked down by an automobile; immediate loss of consciousness; brought to the hospital in the automobile.

Examination upon admission (20 minutes after injury).—Temperature, 97.2°; pulse, 138; respiration, 30; blood-pressure, 112. Semiconscious; in severe shock. Large laceration of 2 inches over right posterior frontal bone; gentle probing reveals simply a crack in the underlying bone. Profuse bleeding from nose; both eyes ecchymotic with bilateral subconjunctival hemorrhages. Pupils dilated and do not react to light. Reflexes cannot be elicited. Fundi negative.

Treatment.—Expectant palliative; vigorous shock measures instituted; scalp laceration cleansed and dressed.

Examination (24 hours after admission).—Temperature, 98.8°; pulse, 110; respiration, 24; blood-pressure, 124. Stuporous and confused mentally; very restless. Both eyes entirely closed from edema. Pupils equal but react sluggishly. Reflexes—knee-jerks exaggerated but equal; double ankle clonus and suggestive right Babinski; abdominal reflexes present and equal. Fundi (Doctor J. A. Kearney)—"optic disks reddish; retinal veins dilated; details of fundus very indistinct—there being a definite blurring of the upper and nasal margins of both optic disks." Lumbar puncture—bloody cerebrospinal fluid under an increased pressure (approximately 12 mm.).

Treatment.—Expectant palliative. (The operation of cranial decompression and drainage was not advised at this time, first, on account of the presence of shock, and secondly, in the hope that the mild increased intracranial pressure would be lessened by natural absorption, making the operation unnecessary.)

Examination (November 22, 1916—5 days after admission).—Temperature, 99.8°; pulse, 74; respiration, 18; blood-pressure, 132. Conscious but very drowsy; complains of severe headache "all over"; when aroused very restless and irritable. Scalp laceration healing *per primam*. Ecchymosis of both orbits less. Definite motor aphasia (incomplete) has developed during past 6 hours. (Patient, parents and grandparents are all right-handed.) Pupils equal and react normally. Reflexes—knee-jerks very much exaggerated but equal; double ankle clonus and double Babinski; abdominal reflexes absent. Fundi—retinal veins dilated; nasal halves of both optic disks entirely blurred. Lumbar puncture—straw-colored cerebrospinal fluid under marked increase of intracranial pressure (approximately 16 mm.).

Treatment.—As the signs of an increased intracranial pressure were becoming more marked, it was thought advisable to lessen this pressure by mechanical means—that is, by a subtemporal decompression, in order that the patient would not only recover life but that the greatest ultimate improvement would occur.

First Operation (6 days after admission).—Left subtemporal decompression and drainage. (The fact that the Babinski reflex appeared first on the right side and that a definite motor aphasia had occurred in a patient who was right-handed and whose parents and grandparents had all been right-handed, indicated that the left cortical hemisphere was possibly more involved than the right cortical hemisphere; also the danger of infection from the laceration of the right scalp was greater in a right subtemporal decompression.) Usual vertical incision, bone removed and no complications. Dura quite tense; upon incising it, straw-colored cerebrospinal fluid spurted several inches, exposing a very "wet" edematous cortex which tended to protrude but did not rupture. Much cerebrospinal fluid escaped, allowing the cortex to become less tense and then to pulsate almost normally. No definite supracortical or cortical clot exposed. Cortex very much congested but no punctate hemorrhages observed. Usual closure with 2 drains of rubber tissue inserted. Duration of operation, 38 minutes.

Post-operative Notes.—Uneventful operative convalescence; decompression area bulged tensely for several days.

Examination (November 29, 1917—12 days after admission and 6 days after operation).—Temperature, 99.6°; pulse, 76; respiration, 18; blood-pressure, 130. No complaints except dull heavy feeling in head—"no real headache." Motor aphasia began to improve immediately after the decompression operation, so that now speech is practically normal except for a slight slurring of polysyllabic words. Operative area bulging slightly; healing *per primam*. Pupils equal and react normally. Reflexes active; double exhaustible ankle clonus with double suggestive Babinski; abdominal reflexes present but depressed, though equal. Fundi—retinal veins dilated; edematous blurring of both nasal margins but only of nasal half of right optic disk (a very interesting observation inasmuch as the subtemporal decompression was performed on the left side). X-ray (Doctor W. H. Stewart)—"depressed area of bone 2 inches in diameter lying over the right posterior frontal bone; line of fracture extends downward into the base" (Fig. 67). (Through an unfortunate oversight, no X-ray had been taken of this patient until this date and the importance of the findings is self-evident; it shows the necessity of röntgenograms in all patients having head injuries.)

Treatment.—The removal of the depressed area of bone advised in order to lessen the danger of future complications.

Second Operation (13 days after admission).—Removal of depressed area of bone; vertical incision of 2 inches over posterior portion of right frontal bone; retractors inserted, exposing a depressed area of one silver dollar in size and to a depth of almost one inch. Bony edges enlarged by small rongeurs and the depressed area of bone extracted. Dura intact and of normal appearance. Usual closure with 2 drains of rubber tissue inserted. Duration of operation, 16 minutes. Uneventful operative recovery.

Examination at discharge (29 days after admission and 16 days after second operation).—Temperature, 98.8°; pulse, 78; respiration, 20; blood-pressure, 132. No complaints except general soreness of head. Operative areas flush with surrounding scalp; decompression site pulsates normally. Pupils equal and react normally. Reflexes—knee-jerks active but equal; bilateral exhaustible ankle clonus and suggestive left Babinski; abdominal reflexes present and equal. Fundi—retinal veins enlarged; slight hazy blurring of nasal margins of both optic disks.

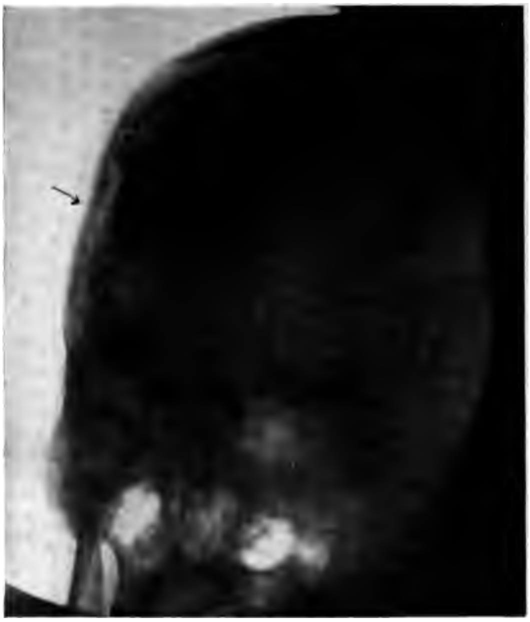

FIG. 67.—An unsuspected extensive and deeply depressed fracture of posterior portion of right frontal bone, disclosed by the X-ray and necessitating its removal for fear of future complications; a left subtemporal decompression had already been performed to lower the increased intracranial pressure. Excellent recovery.

Examination (August 20, 1917—9 months after injury).—No complaints except for dizziness in the morning; also "I've lost my nerve—I can't go faster than 30 miles." No signs of motor aphasia can be elicited. Both operative areas have become depressed. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi negative.

Last Examination (September 12, 1918—22 months after injury).—Patient comes for examination and to receive certificate for draft exemption. No real complaints referable to former head injury. Reflexes negative. Fundi negative. Limited military service advised.

Remarks.—Although in most cases of depressed fracture of the vault of the skull associated with increased intracranial pressure, it is more important to relieve this increased pressure by means of the subtemporal decompression than to elevate or remove the depressed area of the vault, yet the depressed area should always be elevated and removed when possible; the value of careful röntgenograms for patients of this type and I believe for all patients having cranial injuries, is well illustrated in this patient, and it was most important that the depressed fracture of this extent should have been removed, otherwise the great danger of possible future complications. If there had not been localizing signs pointing to the left cerebral cortex as the site of the greater impairment, the decompression operation would naturally have been performed upon the right side and undoubtedly this depression of the vault would then have been ascertained; however, if this depression had been located in another area of the vault, it too would have been overlooked unless careful röntgenograms had been made.

The gradual increase of the intracranial pressure as measured at lumbar puncture and as revealed by the ophthalmoscope is very impressive, and it is these careful and repeated examinations which make it possible for the signs of increased intracranial pressure to be revealed and thus the dangerous compressive effect of extreme intracranial pressure anticipated—both the immediate results and, if death is avoided, then the ultimate remote effects of a prolonged increase of this intracranial pressure.

The disappearance of the paraphasia, following the left subtemporal decompression and the improvement of the reflexes which now pointed to the right cerebral cortex (the site of the depressed area of the vault) as being the greater impaired, was a very interesting observation, and then for all of these signs of cerebral impairment to disappear following the second operation and removal of the depressed area of bone, is worthy of comment.

Acute severe brain injuries associated with a high intracranial pressure due to hemorrhage and to cerebral edema, and requiring the cranial operation of subtemporal decompression.

The presence or not of a fracture of the skull in these patients having a severe brain injury associated with a high intracranial pressure is important only from the standpoint of possible drainage of blood and cerebrospinal fluid through the line or lines of fracture into the ear, nose or subcutaneous tissues of the scalp, and thus a sufficient lowering of the increased intracranial pressure will result so that the operation of decompression and drainage may be avoided. Fortunately, in some of the patients, the base or vault of the skull has been so badly fractured into several movable fragments with profuse drainage that a decompression is not necessary—a sort of natural decompression having been performed; in this manner, the intracranial pressure is relieved to such a degree that the patient recovers to the surprise of all. An ophthalmoscopic and spinal manometric examination in these patients, however, would show that the intracranial pressure had not been markedly increased; this explanation undoubtedly accounts for the recovery of those patients reported as having the most severe forms of comminuted fractures of the skull and yet the recovery is uneventful. In other patients, however, the symptoms and clinical signs at first indicate only a mild type of brain injury with and without a fracture of the skull, and yet the condition of the patient gradually becomes worse, unconsciousness supervenes, and the patient dies with the typical signs of medullary collapse, usually on the fifth or sixth day after the accident, or, in some cases, as late as the fourteenth day; I am confident that if repeated ophthalmoscopic and spinal manometric examinations had been made, the increasing intracranial pressure (apparently "latent" clinically) would have been ascertained and its immediate relief by a cranial decompression and drainage would have saved many of these patients.

Even if the condition of the patient is carefully followed by repeated ophthalmoscopic and the routine neurological examinations, a definite prognosis regarding recovery must be very guarded and naturally should not be given; shock, individual resistance, and the other possible complications are such important factors in each patient that any definite prognosis is most hazardous; in addition to the great danger of medullary compression and, if this danger should be avoided, the possibility of post-traumatic conditions appearing and making the after-life of the patient most pitiable, there is still the ever-present risk of pneumonia occurring in patients confined to their beds in a semiconscious condition and especially is this true of patients over fifty, and if alcoholic to any degree; the danger of pneumonia following an anesthetic, if properly administered, is small in comparison.

If the intracranial pressure in these patients has reached such a height that the expectant palliative method of treatment is no longer considered sufficient to obtain the best results, both immediate and remote, then the mechanical relief of this intracranial pressure by means of the subtemporal decompression and drainage is advisable and at an early date when the condition of the patient is still good; the operation must not be delayed until the patient has reached the severe stage of extreme medullary compression—and surely not that of medullary edema, as it is then too late to obtain a living patient. Upon performing the operation of subtemporal decompression, if it should be found that the intracranial pressure is extreme and especially of the swollen edematous type with very little escape of cerebrospinal fluid and the cerebral cortex pulsates slightly if at all, then a similar decompression should be immediately performed upon the other side of the head—that is, a bilateral decompression. The benefit of this second operation in these selected patients outweighs the added stock of the second operation and this method has proved beneficial in a number of patients. Only about 5 per cent. of the patients requiring the operation of cranial decompression in this series of cases had a bilateral decompression performed and the results were excellent; in this manner, not only was the recovery of life greater, but the ultimate result of the former normality was more frequently obtained than would have been possible following an incomplete relief of the increased intracranial pressure.

It rarely occurs in these patients that the hemorrhage is of such a large amount that it alone is responsible for the height of the intracranial pressure; it is usually due to a subdural hemorrhage associated with a cere
edema of varying degree; of these two factors, the acute condition of cere
edema is probably of greater amount and frequency and therefore of
more importance; the formation of supracortical adhesions and of cy
conditions, however, results from hemorrhage, and therefore its re
effects, unless it is drained early, may be serious, especially when associ
with a varying degree of chronic cerebral edema. At the operation, as m

does not react to light. Reflexes: knee-jerks—right more active than left; exhaustible right ankle clonus and right Babinski; abdominal reflexes—right cannot be obtained. Fundi—definite enlargement of retinal veins; distinct blurring of nasal halves of both optic disks, especially left. Lumbar puncture—blood-tinged cerebrospinal fluid under increasing pressure (approximately 17 mm.).

Treatment.—It was now thought advisable to perform a left subtemporal decompression as the signs of intracranial pressure were increasing.

Operation (31 hours after admission).—Left subtemporal decompression: usual vertical incision, bone removed and no complications; upon retracting the underlying temporal muscle, there was exposed a transverse fracture of the squamous portion of the temporal bone—extending backward and downward; a bony opening of almost 3 inches in diameter was made. Dura very tense and bluish; upon incising it, almost pure blood welled out of dural opening, exposing a supracortical blood clot over 1 c.c. in thickness; this exposed clot was easily removed by salt solution and forceps. Cortex itself edematous and congested but otherwise negative; pulsated normally at end of operation. Usual closure with 2 drains of rubber tissue inserted. Duration of operation, 45 minutes.

Post-operative Notes.—Excellent operative recovery; blood-tinged cerebrospinal fluid continued to drain for 2 days; operative incision healed *per primam.*

Examination at discharge (14 days after admission).—Temperature, 99°; pulse, 76; respiration, 22; blood-pressure, 140. No complaints, but he insists upon leaving the hospital; as patient had no one to take care of him at home, it was thought advisable to transfer him to Bellevue Hospital for convalescence. Site of decompression opening bulging slightly; pulsates normally. Otoscopic examination reveals an almost closed laceration of the posterior portion of left tympanic membrane; bone conduction greater than air conduction in left ear. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi congested but otherwise negative.

Examination (January 10, 1916—30 months after injury).—Patient remained on the Island for 6 weeks; then returned home and has been leading the same life as before the injury—"doing nothing." No complaints, except those due to alcoholism. Hearing negative; air conduction greater than bone conduction. Reflexes negative. Fundi negative.

Last Report (March 10, 1918—57 months after injury).—Letter from brother states: "James died day before yesterday from pneumonia."

Remarks.—The pupillary status of this patient was interesting from the standpoint that the ipsolateral cortical irritation was first exhibited by the contracted left pupil and its sluggish reaction to light; then as the irritative effects of the left supracortical hemorrhage were overshadowed by the paralytic compressive effect of an increasing amount of hemorrhage, then the contracted left pupil gradually enlarged and became dilated and would not react at all to light. This inequality of the pupils in these patients is of great localizing significance; the findings at the left subtemporal decompression confirmed these observations.

The negative fundal findings at the ophthalmoscopic examination upon admission confirmed the presence of initial shock—the temperature then being 97.8; at the examination 30 hours after admission, there were definite signs of pressure in both fundi and particularly the left, and at this examination the temperature had ascended to 99.8° and the blood-pressure to 144, whereas the pulse- and respiration-rates had decreased to 72 and 18, respectively,—definite signs pointing to the overshadowing of the initial shock by an increasing intracranial pressure. It is unfortunate that a second lumbar puncture was not performed at this time, as it would have undoubtedly shown a higher pressure than at the first examination.

It is to be regretted that a post-mortem examination could not have been performed upon this patient and to have ascertained the intracranial condition following an interval of almost 5 years since the brain injury.

The right pupil being larger than the left and apparently dilated more than normally, together with the weakness of the lower portion of the left side of the face, indicated a right cortical involvement; however, it is possible that the left pupil was abnormally contracted due to homolateral cortical irritation, and that the weakness of the lower portion of left side of face was not cortical in type but mildly peripheral in type due to a slight edematous compression of the left facial nerve caused by the proximity of the fracture of the skull producing a laceration of the left tympanic membrane, as revealed by otoscopic examination. A right subtemporal decompression would have been advisable in this case if the reflexes had not been exaggerated on the right side, and if the signs of increased intracranial pressure had been exceedingly high, then it would have been better judgment, in a right-handed individual, to have performed a right subtemporal decompression first, to be followed, if necessary, by a left subtemporal decompression.

The definite temporary recovery obtained in this patient is most gratifying and especially in view of his age of 65 years at the time of the injury. It is indeed surprising how quickly these patients recover following an efficient subtemporal decompression and drainage—even patients having most serious signs, such as convulsive seizures, paralyses and extreme intracranial pressure, provided this pressure can be relieved early—as early as possible following the subsidence of the initial shock.

CASE 43.—Acute severe brain injury without a fracture of the skull; signs of high intracranial pressure associated with subdural and subarachnoid hemorrhage. Left subtemporal decompression and drainage. Excellent recovery.

No. 092.—Hilary. Eighteen years. White. Single. College. U. S.

Admitted March 21, 1914, Polyclinic Hospital. Referred by Doctor T. J. Kearns.

Operation March 21, 1914—5 days after injury. Left subtemporal decompression and drainage.

Discharged March 31, 1914—10 days after operation.

Family history negative; no history of epilepsy, insanity or nervous diseases; no alcoholism.

Personal history negative; 9 months' baby; normal delivery; never had convulsions. Well educated; 5th year Latin; speaks German, French and Greek (even).

Present Illness.—Five days ago (March 16, 1914) while at bat, patient was struck by a pitched ball over left temporo-parietal region; unconscious for several minutes but was unable to finish the game; walked to his home for supper that evening. No bleeding from ears nor nose; no ecchymoses. *Twelve hours later,* patient had an epileptiform attack—25 minutes' duration; its character was not observed other than being a general convulsion with frothing at the mouth; after this attack, a weakness of the right arm was noticed. *Eighteen hours later*—a second convulsion of the Jacksonian type occurred—the spell beginning in the right arm, with frothing at the mouth, then the right side of face, right leg and finally a general convulsion lasting 3 minutes. Since then, patient has had 5 convulsions—the last one being 12 hours ago and was the most severe one—lasting 4 minutes, to be followed by extreme prostration.

Consultation at Patient's Home (March 21, 1914, 4 P.M.—5 days after injury).—Temperature, 99°; pulse, 80; respiration, 16; blood-pressure, 126. Conscious but confused mentally; memory impaired for recent events. Distinct motor aphasia and paraphasia—unable to find the correct word at times, uses words incorrectly and frequently could not repeat single words; patient, however, was conscious of his mistakes. No agraphia. Pupils—left slightly larger than right and reaction to light sluggish. No ocular paralysis. No bleeding from nose, mouth nor ears; no mastoid ecchymosis. Definite weakness of right side of body, particularly of right arm—right-hand grip being much weaker than left. Reflexes—knee-jerks more active on right side; no ankle clonus but a right Babinski; abdominal reflexes—right distinctly depressed. Fundi—retinal veins dilated, nasal margins, particularly of left optic disk, blurred and obscured by edema.

Treatment.—On account of the localized convulsions, the motor aphasia and paraphasia (the patient, his parents and grandparents being all right-handed), the increased reflexes and weakness of the right side, together with the signs of increasing intracranial pressure, the patient was advised to be taken immediately to the hospital and a left subtemporal decompression performed.

Examination upon admission to hospital (March 21, 1914, 9 P.M.).—Temperature, 99.2°; pulse, 78; respiration, 18; blood-pressure, 140. Patient has become more stuporous though no convulsions have occurred since the preceding examination. Paresis of right side of body has increased, especially of right arm and of right side of face. Aphasia and paraphasia have also become more marked, though a careful examination cannot be made on account of the mental impairment. Pupils—left larger than right and reacts sluggishly to light. Reflexes: knee-jerks—right much more exaggerated than left; definite right patellar and right ankle clonus and right Babinski; right abdominal reflex absent. Fundi—nasal halves of both optic disks, particularly the left, obscured by edema.

Treatment.—Left subtemporal decompression and exploration advised.

Operation (March 21, 1914, 10 P.M.—5 days after injury).—Left subtemporal decompression: usual vertical incision (somewhat anterior to usual line), bone removed and no complications; the bone itself, however, was unusually thin in this area but no fracture ascertained. Dura was under high tension and upon incising it, blood-tinged cerebrospinal fluid escaped; upon enlarging dural opening there was exposed a bluish subarachnoid clot—one-fourth inch in thickness and apparently 2 inches in width, extending upward and backward from the anterior portion of the bony opening over the posterior portion of the left third frontal convolution, and then extending backward and upward over the motor area of the face and arm; surrounding cortex edematous but otherwise normal. The arachnoid membrane overlying the hemorrhagic clot was incised, allowing much of the clotted blood to escape, but no attempt was made to remove the entire hemorrhage itself for fear of damaging the underlying cortical cells. Much cerebrospinal fluid escaped, allowing the brain to pulsate normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 50 minutes. Uneventful operative recovery.

Examination at discharge (5 days after injury and 10 days after operation).—Temperature, 98.8°; pulse, 80; respiration, 22; blood-pressure, 128. Speech impairment has practically disappeared, though slight slurring of words occurs when test phrases are used. Weakness of right side of body much improved, but it can still be elicited by the usual tests—hand grip, etc. Decompression area protrudes slightly; normal pulsation. Pupils equal and react normally. Reflexes—right more active than left; no Babinski nor ankle clonus; abdominal reflexes present and equal. Fundi—retinal veins slightly dilated; nasal margins of optic disks, particularly the left, not distinct.

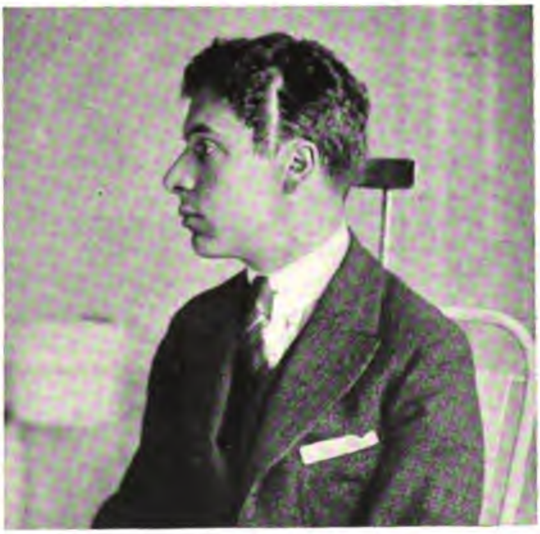

FIG. 68.—The vertical scalp incision of the decompression area exposed by brushing the hair apart—two and one-half years following the operation. No complaints. Excellent recovery.

Examination (June 15, 1914—3 months after injury).—No complaints. No convulsions; no aphasia nor paraphasia elicited. No weakness of right side of body. Decompression area slightly depressed; normal pulsation. Reflexes negative. Fundi negative.

Examination (September 12, 1916—30 months after injury).—No complaints; comes for physical examination before entering law school next month. No aphasia nor paraphasia. No weakness of right side of body. Reflexes negative. Fundi negative. The photograph shows the normal depressed appearance of the decompression area (Fig. 68).

Last Examination (November 6, 1918—56 months after injury).—Patient wishes a certificate of physical well-being in the hope that he may enlist in the army. No complaints. Decompression area depressed; does not pulsate—due possibly to new bone formation. Reflexes negative. Fundi negative. (Patient was admitted to limited service branch of National Army.)

Remarks.—It will be very interesting to follow this patient over a longer period of years to ascertain his complete recovery or not. The danger of possible epileptiform seizures of the minor and major type must be considered in the ultimate prognosis; anyone having convulsions and therefore whose cerebral cortex has once reached a condition of irritability sufficient to result in motor convulsive seizures, is more susceptible to epileptiform spells later, and these patients must be most careful of their habits—especially of diet, which should consist chiefly of non-proteid foods and of small amount, and the total abstinence of alcohol in any form; coffee likewise should be prohibited for these patients. Any unusual strain, mental and physical, should be avoided.

The almost disappearance of the symptoms and signs referable to the left cerebral cortex within 12 hours after the operation was most impressive; the impairment of speech improved so rapidly that it was most surprising to the patient himself, who remarked that "words come now so easily."

It is unfortunate that a lumbar puncture with a measurement of the pressure of the cerebrospinal fluid was not performed upon this patient; the signs of pressure were so definite and the localization so indicative of the left cerebral cortex, especially the Jacksonian convulsive seizures, that the lumbar puncture was not considered necessary. To-day, however, it would be performed in a similar patient, and it should always be performed upon these patients as the most accurate method of determining the intracranial pressure.

CASE 44.—Acute severe brain injury; signs of high intracranial pressure associated with subdural and cortical hemorrhage. Left subtemporal decompression and drainage. Excellent recovery.

No. 280.—David. Twenty-six years. White. Single. Chauffeur. U. S. Admitted June 9, 1915, Polyclinic Hospital.

Operated June 10, 1915—20 hours after injury. Left subtemporal decompression and drainage.

Discharged June 28, 1915—18 days after operation.

Family history negative.

Personal history negative.

Present Illness.—Patient was brought to the hospital by strangers in a taxicab and then abandoned—no history being obtained.

Treatment.—Expectant palliative; scalp laceration widely shaved, cleansed and loosely sutured with 2 drains of rubber tissue inserted.

Examination (18 hours after admission).—Temperature, 100.2°; pulse, 66; respiration, 16; blood-pressure, 140. Patient conscious but confused mentally and badly oriented. Definite paraphasia present. Pupils—left widely dilated and does not react to light. Reflexes: knee-jerks—right greater than left; right ankle clonus and right Babinski; abdominal reflexes increased—left more than right. Fundi—both optic disks blurred by edema but no measurable papilledema; retinal veins dilated and tortuous. Lumbar puncture—bloody cerebrospinal fluid under high pressure (approximately 16 mm.). No definite weakness of right side of body elicited.

Treatment.—Left subtemporal decompression advised for fear the increasing intracranial pressure would produce an extreme medullary compression and thus possibly precipitate a medullary edema; it was thought advisable to operate now rather than to wait until the patient might enter a far more serious condition of medullary compression, when it would be very doubtful if the patient would recover life itself.

Operation (June 12, 1915—20 hours after admission).—Left subtemporal decompression: usual vertical incision, bone removed and no complications; no fracture of bone ascertained in this area. Dura tense and bluish; upon incising it, bloody cerebrospinal fluid spurted to a height of 5 inches, and upon enlarging the dural opening a very "wet" edematous and congested cortex with numerous punctate hemorrhages in it tended to protrude under high tension; the cerebral tension was so high that a small rupture of the temporal lobe beneath the Sylvian fissure occurred. Much blood and cerebrospinal fluid escaped so that the brain pulsated at the end of the operation. No large clots ascertained. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Uneventful operative recovery, although patient was irrational for 3 days following the operation, and he was mentally confused for almost 10 days after operation—getting out of bed and insisting upon going home; incision healed *per primam.*

Examination at discharge (19 days after admission and 18 days after operation).—Temperature, 99°; pulse, 70; respiration, 20; blood-pressure, 134. No complaints. Patient unusually restless and excitable and does not sleep well. Decompression opening bulges under high pressure; pulsation normal. Pupils equal and react normally. Hearing impaired in right ear; otoscopic examination reveals a small laceration of posterior portion of right tympanic membrane. Reflexes: active but otherwise negative; abdominal reflexes increased but equal. Fundi—nasal margins of both optic disks obscured by edema but nasal halves and temporal margins are clear and distinct; retinal veins enlarged. X-ray (Doctor A. J. Quimby)—"negative for fracture of the skull."

Examination (January 10, 1917—18 months after injury).—No complaints; works daily. Decompression area depressed and pulsates normally. No impairment of hearing of right ear; air conduction greater than bone conduction. Reflexes: active but otherwise negative; abdominal reflexes active but equal. Fundi negative.

Last Examination (September 10, 1918—39 months after injury).—Patient comes to see me before embarkation with the army. No complaints. Otoscopic examination reveals a normal right tympanic membrane; hearing of right ear apparently normal (rough tests of watch, etc.). Reflexes active but otherwise negative. Fundi negative. Decompression area depressed and apparently being filled in with new bone formation.

Remarks.—Although the fracture in this patient was undoubtedly on the right side, causing a rupture of the right tympanic membrane, yet a *left* subtemporal decompression was performed because the signs of cortical involvement indicated a left intracranial lesion beside the general increased intracranial pressure; as is well known and as this case illustrates, the fracture of the skull is possibly the least important factor to be considered in intracranial injuries. It is possible that the fracture extending through the right ear permitted the intracranial blood over the right hemisphere to escape through the right auditory canal and thus there were no signs of a lesion of the right hemisphere, whereas the left hemisphere not having any outlet for its supracortical "free" blood exhibited the signs of its irritation and impairment—paraphasia (the patient being right-handed), dilated left pupil and non-reacting to light, increased deep reflexes on the right side in addition to the right ankle clonus and right Babinski while the right abdominal reflexes were absent. Thus the right cerebral hemisphere may have been "decompressed" and drained through the right ear, and the left cerebral hemisphere not being "decompressed" (to the extent of the right hemisphere) gave the greater evidence of impairment—beside the signs of a general increase of the intracranial pressure.

The very early return to normal function of the right ear within a period of 18 months is very impressive; naturally, this excellent result is only possible in the absence of middle ear infection following the laceration of the tympanic membrane, and when the line of fracture has in no way permanently damaged the transmitting mechanism of the tympanic ossicles or of the internal ear and auditory nerve itself; these latter impairments are usually permanent.

To make the otoscopic examination at the time of the patient's admission to the hospital and in the presence of a profuse discharge of blood from either ear, is a distinctly dangerous procedure and should never be undertaken for fear of introducing infection into the middle ear by the swabbing out of the external auditory canal in order to obtain a clear view of the tympanic membrane; many cases of local and general meningitis with or without the complication of brain abscess are frequently produced in this manner; it is always wiser to wait until the discharge of blood and cerebrospinal fluid has ceased before an otoscopic examination is made. In many patients the rapidly increasing signs of the intracranial pressure are undoubtedly due to cerebral edema rather than to an intracranial hemorrhage itself; the "natural" decompression by means of drainage through a ruptured tympanic membrane is not always sufficient to lessen markedly the increasing intracranial pressure; many patients, however, are thus spared an operation by means of this sort of "natural" decompression.

A measurement of the pressure of the cerebrospinal fluid at lumbar puncture should always be performed upon these patients, as we now know that it is a much more accurate and delicate test of intracranial pressure than the ophthalmoscopic examinations of the fundi; even in conditions of mild initial shock and where the fundi are negative, yet it is frequently possible to find a pressure of the cerebrospinal fluid of approximately 12 and even 14 mm.; a second lumbar puncture later upon many of these patients would undoubtedly register a much higher pressure.

This patient is a good illustration of a serious brain injury and yet no fracture of the skull could be demonstrated; the fracture may have been present but it in itself is not an important factor in the diagnosis and treatment of brain injuries; the röntgenogram was not taken until the patient was well enough to be discharged and then merely as a question of record and for fear that a depressed fracture of the vault might be overlooked. No cranial operation to relieve the increased intracranial pressure in these patients should be delayed merely in order to obtain an X-ray picture of the skull; the operation is not performed for the fracture nor on account of the fracture.

The early fundal signs of increased intracranial pressure revealed by the ophthalmoscope within one hour after the injury can occur so quickly following cranial injuries only in the absence of shock, or as the result of a large hemorrhage occurring immediately following the cranial injury, and even in this latter condition the consequent shock would tend to lessen this increased intracranial pressure.

It is most unusual in these patients having cranial injuries to demonstrate increased abdominal reflexes, as the usually associated factor of shock tends to abolish them, and if shock is not present in a marked degree but an increased intracranial pressure, then this latter factor tends to depress the abdominal reflexes; in fact, if the abdominal reflexes are not equal, the depressed ones are opposite the side of the greater cortical compression—that is, one cerebral hemisphere more seriously impaired and compressed produces on the opposite side of the body increased deep reflexes but diminished superficial reflexes, such as the abdominal reflexes. It was, therefore, very surprising to elicit increased abdominal reflexes in this patient before operation, and they remained increased at every examination subsequent to the operation.

Case 45.—Acute severe brain injury; signs of a high intracranial pressure associated with subdural and subarachnoid hemorrhage. Repeated lumbar punctures. Left subtemporal decompression and drainage. Excellent recovery.

No. 283.—Gustave. Twenty-eight years. White. Single. Elevatorman. U. S.

Admitted June 10, 1915, Polyclinic Hospital.

Operation June 18, 1915—8 days after injury. Left subtemporal decompression and drainage.

Discharged July 18, 1915—30 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While working in a department store, patient fell a distance of one story down the elevator shaft; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (1 hour after injury).—Temperature, 97.6°; pulse, 94; respiration, 28; blood-pressure, 118. Semiconscious; in moderate degree of shock. Profuse bleeding from left ear; left mastoid ecchymosis. Pupils slightly dilated but react to light normally. Reflexes obtained with difficulty but otherwise negative. Fundi negative.

Treatment.—Expectant palliative; vigorous shock measures instituted. Within 12 hours patient recovered from the condition of shock, became more conscious, complaining of severe headache, and after 48 hours, patient became more and more drowsy; slept for long periods of time and began to show definite signs of an increased intracranial pressure. Repeated lumbar punctures were performed on six successive days and 15–20 c.c. of clear cerebrospinal fluid were removed each time under high pressure (approximately 14–17 mm.); the last puncture and withdrawal of fluid was on June 18, 1915.

Examination (June 18, 1915—8 days after admission).—Temperature, 101.2°; pulse, 66; respiration, 16; blood-pressure, 138. Conscious but confused mentally; complains of severe headache and "I want to go home." Bleeding from left ear has ceased; otoscopic examination reveals a large laceration of posterior portion of left tympanic membrane. No aphasia nor weakness of right side of body. Pupils equal and react normally. Reflexes: knee-jerks—right exaggerated; right exhaustible ankle clonus and right Babinski; abdominal reflexes present and equal. Fundi—retinal veins markedly dilated; both optic disks blurred with edema, though their temporal margins can still be observed and therefore no measurable swelling or papilledema. Lumbar puncture—clear cerebrospinal fluid but under very high pressure (approximately 20 mm.).

Treatment.—A left subtemporal decompression and drainage was now advised in the belief that the patient would not be able to absorb the cerebral edema normally and in the knowledge that the intracranial pressure was daily becoming higher and thus the danger of an acute medullary compression and edema was to be feared,—therefore it should be anticipated and prevented if possible.

Operation (June 18, 1915—8 days after admission).—Left subtemporal decompression: usual vertical incision, bone removed and no complications. Dura very tense and bluish in areas; upon incising it, bloody cerebrospinal fluid spurted to a height of 8 inches and continued for at least five seconds. Upon enlarging dural opening, much bloody cerebrospinal fluid and many dark blood clots extruded. The "wet" edematous cortex tended to protrude and finally ruptured at one point beneath the Sylvian fissure; this cerebral tension was so great that a ventricle puncture was performed but only a small amount of clear cerebrospinal fluid escaped —therefore, the intracranial pressure was due to cerebral edema and to hemorrhage rather than to a blockage of the ventricles. A large amount of bloody cerebrospinal fluid escaped but the cortex was still bulging at the end of the operation. (A bilateral decompression and drainage was considered but it was finally decided to wait in the hope that this one decompression would be sufficient to lessen the intracranial pressure satisfactorily.) Usual closure with 2 drains of rubber tissue inserted. Duration, 65 minutes.

Post-operative Notes.—Uneventful operative recovery, although for 3 weeks following the operation the patient was in a confused mental state so that it was necessary to have him closely watched; incision healed *per primam.*

Examination at discharge (38 days after admission and 30 days after operation).—Temperature, 98.6° ; pulse, 74; respiration, 18; blood-pressure, 130. Perfectly conscious and rational now, though patient is in a rather unstable condition emotionally, saying, "I am so homesick and sad." Decompression wound bulging but not so tense as one week ago when the patient was slightly irrational. Impairment of hearing of left ear—bone conduction being greater than air conduction. Pupils equal and react normally. Reflexes very active but otherwise negative. Fundi—retinal veins enlarged; nasal margins of both optic disks indistinct and obscured by edema. X-ray (Doctor A. J. Quimby)—"bony defect only of decompression demonstrated" (Fig 69).

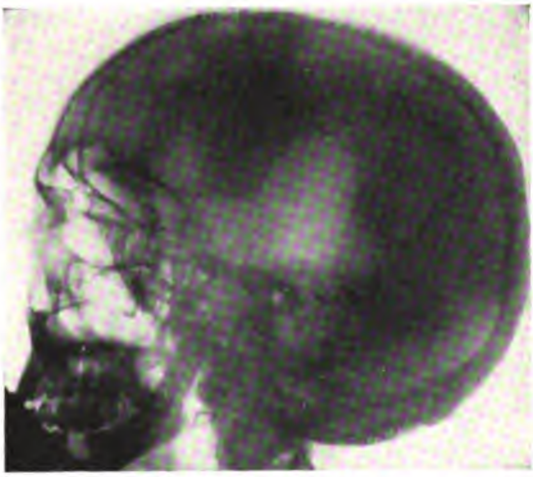
FIG. 69.—Oval bony defect of a left subtemporal decompression in a patient having a high intracranial pressure due to subdural and subarachnoid hemorrhages. Excellent recovery.

Treatment.—Patient was advised to remain at home quietly and not to work for a period of at least 3 months.

Examination (September 10, 1916—15 months after injury).—No complaints; works daily in his old position. Decompression area depressed; pulsates normally. Hearing of left ear less acute than right; bone conduction equals air conduction. Reflexes active but otherwise negative. Fundi negative.

Last Examination (September 22, 1918—39 months after injury).—No complaints and is still working in the same place. No impairment of hearing of the left ear can be ascertained; otoscopic examination negative. Reflexes active but otherwise negative. Fundi negative. The operative area is "sunken in," as shown by the photograph (Fig. 70).

Remarks.—It would undoubtedly have been better surgical judgment to have operated earlier upon this patient, but it was hoped after the shock of the injury had disappeared that the patient could recover without an operation being necessary; it was doubted at the time of the operation if the patient would entirely recover ultimately—that is, be as normal an individual after the injury as before it; apparently he is going to recover completely. It was interesting to observe that as long as the intracranial pressure remained high so that the decompression area was bulging and tense, just so long was the patient slightly irrational and in a confused state mentally and emotionally; when this intracranial pressure became less, then the mental condition and emotional instability immediately improved. Another point worthy of note is the fact that clear cerebrospinal fluid was obtained at lumbar puncture seven different times before the operation, and yet at operation there were found bloody cerebrospinal fluid and many small subdural blood-clots; as has been observed frequently in other patients of this series, clear cerebrospinal fluid at lumbar puncture does not rule out the possibility of subdural hemorrhage; in these patients, this may be due to a blockage of the subarachnoid space at the foramen magnum, but this seems very improbable as the danger of a medullary compression would then be very great indeed.

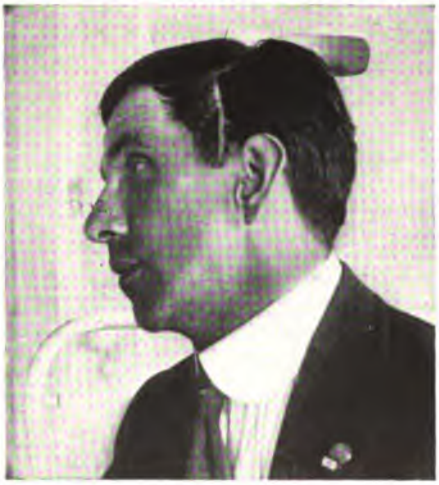
FIG. 70.—Vertical scar of left subtemporal decompression (exposed by brushing apart the hair) three years after the operation. The depression of the operative area indicates the complete lowering of the intracranial pressure to normal.

The operation was also delayed for several days longer than it should have been in the belief and hope that the repeated daily lumbar punctures and withdrawal of 15 to 20 c.c. of cerebrospinal fluid each time would so lessen the increased intracranial pressure that it would be unnecessary to perform a subtemporal decompression and drainage; the relief, however, was a temporary one—not lasting longer than 6 to 8 hours after the withdrawal of the fluid, and it is a mistake in these patients having high intracranial pressure to attempt to lower it by the method of lumbar puncture; besides, there is a definite danger of inducing an acute medullary compression in these patients having high intracranial pressure—if over 15 mm., as registered by the spinal mercurial manometer. After it was ascertained that the intradural pressure was very high and upon making a small opening in the dura the underlying cortex had tended to protrude, it would have been better surgical judgment if then, through this small dural opening a ventricle puncture needle had been used to tap the ventricle and in this manner the cerebrospinal fluid had been permitted to escape; thus, the intradural pressure would have been so lessened that it would have been possible to enlarge the dural opening widely and there would not have been any danger of the underlying cortex being damaged or ruptured, as, unfortunately, occurred in this patient; it being the temporo-sphenoidal lobe, however, naturally there appeared no clinical signs of its presence.

CASE 46.—Acute severe brain injury; signs of an increasingly high intracranial pressure associated with subdural and cortical hemorrhage. Right subtemporal decompression and drainage. Excellent recovery.

No. 526.—Joseph. Thirty-six years. White. Single. Laborer. Italy. Admitted March 1, 1917, Polyclinic Hospital.

Operation March 8, 1917—7 days after injury. Right subtemporal decompression.

Discharged April 4, 1917—27 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While crossing the street, patient was struck by an automobile; immediate loss of consciousness; brought to the hospital in the automobile.

Examination upon admission (20 minutes after injury).—Temperature, 97.4°; pulse, 110; respiration, 32; blood-pressure, 120. Semiconscious and in severe shock. Profuse bleeding from left ear; extensive ecchymosis of left mastoid area. On account of the severe shock, patient was not examined thoroughly at this time.

Treatment.—Expectant palliative; vigorous anti-shock measures instituted.

Examination (48 hours after injury).—Temperature 99.2°; pulse, 88; respiration, 24; blood-pressure, 128. Definite general improvement. Stuporous, but when aroused very restless and irritable. Bleeding from left ear has ceased; otoscopic examination reveals an extensive laceration of entire posterior portion of left tympanic membrane. Pupils—right contracted and much smaller than left; sluggish reaction to light. Reflexes—knee-jerks exaggerated, left greater than right; suggestive left Babinski; abdominal reflexes both depressed, left possibly more than right. Fundi—retinal veins enlarged with distinct blurring of nasal margins of both optic disks. Lumbar puncture could not be performed as patient was so restless and difficult to manage that it was feared the needle might be broken unless an anesthetic was administered, and this was not thought advisable.

Treatment.—Expectant palliative; patient seemed to improve during the following six days.

Examination (7 days after admission).—Temperature. 100.4°; pulse, 52; respiration, 14; blood-pressure, 134. Patient has become more stuporous during last 24 hours. Pupils equal and react normally, though slightly sluggish; at times, right pupil much smaller than left. Reflexes—knee-jerks exaggerated, left more than right; left Babinski; abdominal reflexes—left can scarcely be obtained. Fundi—retinal veins tortuous and engorged; definite papilledema of both disks—edematous blurring and obscuration of both nasal and temporal halves of optic disks having a measurable swelling of 3 D.—that is, the condition of "choked disks." Lumbar puncture—bloody cerebrospinal fluid under extreme pressure (45 mm.)!!

Treatment.—An immediate right subtemporal decompression advised at this time as the signs of increasing intracranial pressure were becoming more and more extreme, and thus showing that the patient could not absorb the intracranial hemorrhage and edema by the natural means of absorption.

Operation (7 days after admission).—Right subtemporal decompression: usual incision, bone removed and no complications. Dura very tense and bluish; upon incising it, free blood spurted under high pressure, revealing a very "wet," tense, swollen cortex in which there were numerous punctate hemorrhages. Cortex protruded, but owing to the rapid loss of cerebrospinal fluid it receded before the end of the operation and pulsated almost normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 45 minutes.

Post-operative Notes.—Pulse was 100 at end of operation, but it became 86 within two hours; it was noticed at this time that the left pupil was markedly contracted and very much smaller than right, and it was feared that possibly a hemorrhage had also occurred over the left hemisphere; he was, therefore, carefully watched during the day for any developing signs, but within 10 hours after operation the pupils were equal, the pulse was 72, and there were no signs of paralysis nor convulsions; mentality was also clear. Incision healed *per primam.*

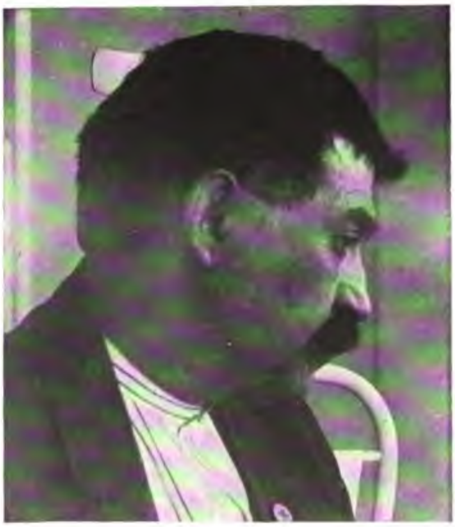

FIG. 71.—Eighteen months after a right subtemporal decompression to relieve an extreme intracranial pressure of 45 mm., as registered by the spinal mercurial manometer and due to an extensive subdural hemorrhage and an extreme cerebral edema. Recovery has been excellent.

Examination at discharge (34 days after injury and 27 days after operation).—Temperature, 99°; pulse, 76; respiration, 20; blood-pressure, 140. No complaints except slight dizziness and general weakness. Definite impairment of hearing of left ear can be demonstrated objectively, but patient is apparently not aware of it; otoscopic examination reveals a large tear of the posterior portion of left tympanic membrane; bone conduction greater than air conduction; slight left mastoid ecchymosis persists. Decompression wound has entirely healed; bulges slightly and pulsates normally. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—retinal veins enlarged; slight edematous haziness along the nasal margins of both optic disks.

Examination (September 10, 1917—6 months after injury).—No complaints; works daily. Decompression area slightly depressed; normal pulsation. Reflexes negative. Fundi negative. Hearing of left ear still impaired—bone conduction being greater than air conduction.

Last Examination (September 22, 1918—18 months after injury).—Occasional headache at times, but feels well and strong; "cannot drink any more as I become intoxicated very easily." Decompression site sunken in; slight pulsation still observed (Fig. 71). Reflexes negative. Fundi negative. Hearing of left ear impaired; bone conduction greater than air conduction; otoscopic examination reveals small opening in posterior half of left tympanic membrane.

Remarks.—At the time of the operation it was thought that the decompression should have been performed 2 or 3 days earlier at least, and it was feared that the patient might not regain his normal condition mentally and emotionally as before the injury; it would undoubtedly have been wiser to have operated earlier, and yet it was hoped that by waiting, the operation would not be necessary; instead of the intracranial pressure being lessened by the expectant palliative method, the signs of increasing pressure became more and more marked, particularly in the fundi, and when this observation was confirmed more accurately by a measurement of the pressure of the cerebrospinal fluid at lumbar puncture, it was forcefully emphasized that no further delay should be permitted and the operation of subtemporal decompression and drainage was immediately performed. The numerous punctate hemorrhages throughout the cortex made me feel at the time of the operation that this patient could never regain his former normality—it seemed incredible that these small cortical hemorrhages would not damage the patient and impair him both mentally, physically, and especially emotionally; and yet one and a half years following the operation, the patient is practically a normal man—possibly a little less stable emotionally, but otherwise well. The excellent recoveries obtained in similar patients who have been treated by the same method would indicate that the brain is capable of absorbing small punctate hemorrhages with little or no ultimate impairment—at least to be demonstrated clinically, and that this is possible chiefly because the increased intracranial pressure has been lowered by the operation which facilitates the natural absorption of these cortical hemorrhages. It is remarkable how patients having so many small hemorrhages in the cortex that the brain has almost the appearance of liver or spleen tissue at operation, and yet the ultimate recovery may be excellent, although usually associated with a definite emotional instability in these severe cases.

The almost immediate disappearance, following operation, of the contracted right pupil and the increased deep reflexes of the left side, including a suggestive left Babinski, illustrates the effectiveness of the right subtemporal decompression and drainage in diminishing the right cortical irritative and compressive factors; a left subtemporal decompression would have been performed immediately after the first operation if the signs pointing to a left cortical lesion had not quickly disappeared. It is only in rare cases of brain injuries that a bilateral decompression and drainage is necessary, and that can be usually decided at the time of the first operation, although in some doubtful cases it may be necessary to wait a period of hours or even days; naturally, if the one operation is considered sufficient for the relief of the increased intracranial pressure, the second operation should be avoided if possible. As in this patient having an extreme intracranial pressure, even if the signs had pointed to a lesion of the left hemisphere, yet it would have been better surgical judgment to have performed a right decompression first and then if necessary a left decompression later—thereby avoiding cerebral damage.

The ultimate recovery of the hearing is the usual result in these patients when the impairment is limited to the middle ear alone and chiefly to one of laceration of the tympanic membrane; these lacerations usually heal within the period of one year at most when the air conduction quickly equals that of bone conduction, and within several months the air conduction is greater than bone conduction, as it normally should be. It is only when the ossicular transmitting mechanism of the middle ear is damaged by an adjacent fracture or the internal ear or the auditory nerve itself is impaired that the marked loss of hearing is permanent. Although no cerebrospinal fluid is observed in the discharge of the ear, yet there is frequently a fracture of the adjacent bone, and I think this is true of many patients where only blood is discharged from the ear—a fracture of the skull is present, although this opinion cannot be stated with certainty at the time unless confirmed by röntgenograms, operation or autopsy.

Case 47.—Acute severe brain injury; signs of high intracranial pressure associated with extradural and subdural hemorrhage with brain laceration. Left subtemporal decompression and drainage. Excellent recovery.

No. 776.—John. Thirty-three years. White. Single. Barkeeper. U. S. Admitted February 11, 1917, Polyclinic Hospital.

Operation February 19, 1917—8 days after injury. Left subtemporal decompression and drainage.

Discharged March 19, 1917—30 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While walking along the pavement near the gutter, patient was struck by an automobile which had run "amuck"; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (35 minutes after injury).—Temperature, 99.4°; pulse, 66; respiration, 16; blood-pressure, 142. Semiconscious and could be aroused by firm supra-orbital pressure; vomited profusely during the examination—almost of projectile type. Extensive hematoma over left parietal bone. No bleeding from nose, mouth or ears; no mastoid ecchymoses. Pupils—left pupil dilated and reacts sluggishly to light. Reflexes—knee-jerks exaggerated but equal; no ankle clonus nor Babinski; abdominal reflexes depressed but equal. Fundi—retinal veins enlarged; nasal margins of both optic disks blurred and indistinct, while nasal half of left optic disk slightly obscured by edema. Lumbar puncture—bloody cerebrospinal fluid under an increased pressure (14 mm.); 2 ounces were allowed to escape slowly and under careful observation of the pulse.

Treatment.—Expectant palliative; it was hoped that the patient would be able "to take care of" this increased intracranial pressure by the natural means of absorption and thus an operation be avoided.

Examination (7 days after admission).—Temperature, 100.6°; pulse, 62; respiration, 16; blood-pressure, 144. Patient not so stuporous as upon admission, but the signs of an increasing intracranial pressure have become more marked. Patient complains continuously of severe headache when he is awake, although he is sleeping most of the time. Pupils—left still larger than right and reacts sluggishly to light. Reflexes: knee-jerks exaggerated—right more than left; double ankle clonus and double Babinski; abdominal reflexes cannot be elicited. Fundi: retinal veins very much dilated and tortuous; outlines of both optic disks hazy and obscured and both nasal and temporal halves blurred by edema—that is, papilledema in its early stages but not of a measurable swelling to the degree of "choked disks." Lumbar puncture—bloody cerebrospinal fluid under a high intracranial pressure (20 mm.).

Treatment.—An immediate left subtemporal decompression advised.

Operation (7 days after admission).—Left subtemporal decompression: usual incision, bone removed and no complications; in the temporal muscle beneath the temporal fascia, there was much free blood and upon retracting the muscle an oblique linear fracture extending downward through the middle portion of the squamous bone was exposed. Upon rongeuring away the bone, a large extradural blood-clot of one-half inch in thickness was extruded through the bony opening; posteriorly, the dura was depressed from the bone to a depth of one inch by this blood-clot, which had an extent of at least 5 inches. This extradural hemorrhage being evacuated, the bluish dura was now incised, allowing considerable subdural hemorrhage to escape and also a small quantity of hemorrhagic brain tissue to be extruded under high pressure; the cortex had been lacerated just beneath the Sylvian fissure; the adjacent brain tissue was hemorrhagic and had almost the appearance of liver tissue. On account of the escape of a large amount of cerebrospinal fluid and blood, the brain now receded and pulsated normally. Usual closure with 2 drains of rubber tissue inserted subdurally, and one gauze tape extradurally toward the left mastoid area, which was bleeding profusely as if the lateral sinus had been torn; this packing was sufficient to stop the bleeding extradurally. Duration, 70 minutes.

Post-operative Notes.—Uneventful operative recovery; patient became conscious within 18 hours and made an excellent convalescence; incision healed *per primam.*

Examination at discharge (38 days after injury and 30 days after operation).—Temperature, 98.6°; pulse, 70; respiration, 20; blood-pressure, 136. Perfectly conscious and rational; sleeps at least 14 hours a day. No complaints other than soreness over left side of head. Decompression area flush with surrounding scalp; pulsates normally. Pupils equal and react normally. Reflexes very active but otherwise negative. Fundi—retinal veins enlarged; nasal margins indistinct and hazy but the other details of both optic disks are clear. X-ray (Doctor W. H. Stewart)—"oval bony defect of left decompression shown; oblique fracture of left vault observed" (Fig. 72).

Examination (November 6, 1917—9 months after injury).—No complaints other than an occasional headache, "after tending bar all day." Reflexes active but otherwise negative. Fundi negative. Decompression area slightly depressed but not so much as it should be; pulsation normal.

Last Examination (September 10, 1918—19 months after injury).—No complaints. Decompression site, however, is only slightly depressed; pulsates normally. Reflexes very active but otherwise negative. Fundi negative.

Remarks.—It would have been better surgical judgment to have advised the decompression at least 2 days earlier, but it was hoped that an operation would not be necessary, and it was not until a definite papilledema appeared and the pressure of the cerebrospinal fluid reached 20 mm. that a decompression was considered obligatory.

It would have been better surgical judgment when the intradural pressure was ascertained to be extreme after a small dural opening had been made, if a lumbar puncture had now been performed and a large amount of cerebrospinal fluid carefully withdrawn; in this manner the intracranial pressure could have been greatly lessened so that the dural opening could be widely enlarged with little or no danger of the underlying cerebral cortex being forced upward through the dural opening and its being ruptured. Although no clinical signs of this laceration have appeared in this patient following the operation (the affected underlying cortex being the temporosphenoidal lobe which is a comparatively silent area of the brain, and especially the right one), still it is inconceivable that some impairment has not occurred, mental or psychical at least, and it is only on account of our crude methods of examination that these slight impairments cannot be ascertained.

The dilated left pupil with sluggish reaction to light was indicative of an ipsolateral compressed left cerebral cortex, and being associated with increased deep reflexes of the right side and possibly greater signs of pressure in the left fundus, this opinion was greatly confirmed and then proven at operation.

Although this patient has apparently entirely recovered his former good health, yet it would undoubtedly have been wiser to have operated several days before in order to assure a greater recovery of normal function—mentally, physically and emotionally, and also to have lessened the danger of an acute medullary compression and even edema itself. This is the type of patient frequently observed who can endure the increased intracranial pressure for a number of days and even several weeks, and then finally and suddenly succumbs to an acute medullary compression and edema. The value of careful, daily and even hourly, examinations of the fundi and also but of less value, of the pulse, respiration and blood-pressure and then the most accurate means of ascertaining the intracranial pressure by means of the spinal mercurial manometer should be utilized, and thus the onset of medullary compression be avoided—as it usually can be in these patients.

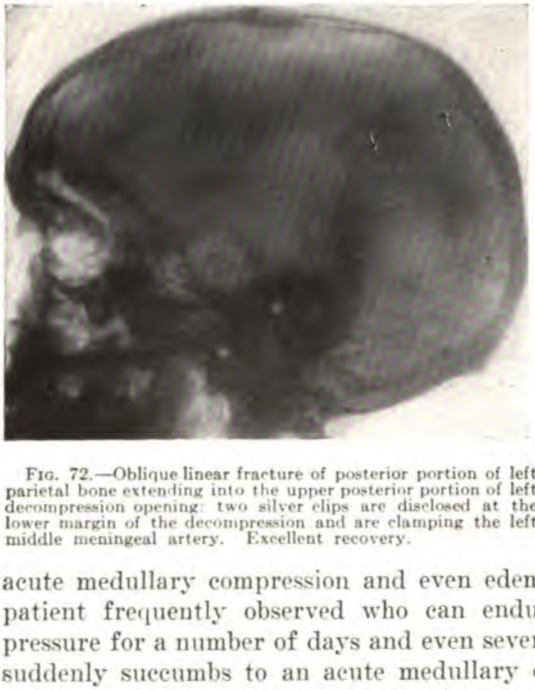

FIG. 72.—Oblique linear fracture of posterior portion of left parietal bone extending into the upper posterior portion of left decompression opening: two silver clips are disclosed at the lower margin of the decompression and are clamping the left middle meningeal artery. Excellent recovery.

B. *Bilateral decompression.*

CASE 48.—Acute severe brain injury; signs of extreme intracranial pressure associated with subdural and intracerebral hemorrhages. Bilateral decompression and drainage. Excellent recovery.

No. 048.—Frank. Thirty-two years. White. Married. Mechanic. U. S.

Admitted March 5, 1913, Polyclinic Hospital. Referred by Doctor W. S. Bainbridge.

Operations March 6 and 7, 1913—33 and 59 hours, respectively, after injury. Bilateral decompression and drainage.

Discharged April 12, 1913—25 days after second operation.

Family history negative.

Personal history negative.

Present Illness.—While crossing the street, patient was struck by an automobile; unconscious for several minutes but was then able to arise and walk to the curb, where he sat down; several minutes later a policeman arrested him as a disorderly character; taken to the station house, and as the patient was unable to talk, he was designated a "foreigner" on the register. Four hours later, he was found semiconscious in his cell and was then transferred to the hospital in the ambulance.

Examination upon admission (5 hours after injury).—Temperature, 101°; pulse, 68; respiration, 18; blood-pressure, 150. Well-developed and nourished. Semiconscious but can be aroused easily by supraorbital pressure. Motor aphasia complete—unable to speak or utter a sound. Bleeding from nose; both orbits ecchymosed—right more than left. Bleeding profusely from left ear; left mastoid ecchymosis. Pupils contracted equally and react to light normally. Reflexes: patellar—right greater than left; right Babinski; right abdominal reflex could not be elicited. Fundi—retinal veins full; nasal margins blurred with edema but temporal margins and nasal halves clear and distinct. Lumbar puncture—bloody cerebrospinal fluid under increased pressure (approximately 14 mm.).

Treatment.—Expectant palliative.

Examination (24 hours after admission).—Temperature, 100.4°; pulse, 62; respiration, 16; blood-pressure, 160. Patient remains semiconscious; unable to speak a word. Physical condition practically the same as at the preceding examination, except that the ophthalmoscope reveals the retinal veins dilated and the nasal halves of both optic disks obscured by edema. Lumbar puncture—bloody cerebrospinal fluid under increased pressure (approximately 18 mm.).

Treatment.—A left subtemporal decompression now advised in order to prevent the onset of a definite medullary compression, and also to expose the motor speech centre in the posterior portion of the third left frontal convolution through the anterior portion of the left decompression opening. (Patient being right-handed—his parents and grandparents also being right-handed—therefore his motor speech centre was in the left cerebral cortex.)

First Operation (March 6, 1913—28 hours after admission).—Left subtemporal decompression: usual vertical incision, bone removed and no complications; much free blood among the fibres of temporal muscle beneath the temporal fascia, therefore a fracture of the underlying bone, and this observation was confirmed by exposing 2 irregular lines of fracture in the squamous portion of the temporal bone. Dura very tense and bluish; upon incising it large dark clots, the size of olives, welled up through dural opening, and upon enlarging dural opening, these dark clots could be seen protruding through a laceration of the posterior portion of the third left frontal convolution—that is, the motor speech area. Brain very tense and "water-logged" with many punctate hemorrhages throughout the cortex, which tended to protrude; as very little cerebrospinal fluid escaped, the cortex, which continued to remain tense, scarcely pulsated, so that by the end of the operation the brain had not receded as it usually does. Usual closure with 2 drains of rubber tissue inserted. Duration, 1 hour.

Post-operative Notes.—There was little or no improvement in the condition of the patient within 24 hours after operation; he remained in a semiconscious condition with a pulse of 62 and a respiration of 16, and as the decompression area was so tense and bulging that it did not pulsate, it was decided to perform a right subtemporal decompression.

Second Operation (26 hours after first operation and 54 hours after admission).—Right subtemporal decompression: usual vertical incision, bone removed and no complications. Dura very tense and only slightly bluish; upon incising it, merely blood-tinged cerebrospinal fluid escaped, allowing the "wet" edematous cortex to pulsate. No subdural clots found, but at the end of the operation as the result of the escape of much blood-tinged cerebrospinal fluid, the cortex lessened its protrusion and pulsated normally. Usual closure with 2 drains of rubber tissue inserted. The left subtemporal decompression area remained so tense and bulging that it was thought advisable to reopen the incision, which was done, and a large subdural clot welled through the dural opening and also small clots, the size of cherries, were extruded through the cortex of the motor speech area; upon removing these clots, the cortex became less tense and the incision was now closed in usual manner with 2 drains of rubber tissue inserted. Duration, 80 minutes.

Post-operative Notes.—On the second day, patient was still unable to talk, but he was conscious and would shake hands with those whom he recognized as his friends. Hearing apparently impaired. Pupils equal and react normally. Reflexes—patellar equally exaggerated and a suggestive double Babinski. Fundi—retinal veins dilated while the nasal margins of both optic disks are still obscured. On the fifteenth day after operation, patient was able to say several words intelligibly.

Examination at discharge (27 days after admission and 26 days after first operation).—Temperature, 98.6°; pulse, 70; respiration, 18; blood-pressure, 142. Perfectly conscious with no complaints; can talk fairly well in a hesitating manner. Hearing of left ear impaired—bone conduction being greater than air conduction; otoscopic examination reveals a laceration of the left tympanic membrane through its inferior portion. Pupils equal and react normally. Reflexes—very active but otherise negative. Fundi—retinal vessels slightly enlarged but no edema of the optic disks. Decompression areas tense but not bulging. X-ray (Doctor A. J. Quimby)—"the bilateral decompression openings are clearly demonstrated" (Fig. 73).

Examination (June 7, 1914—3 months after injury).—No complaints; patient went to work 41 days after operation and is now working daily as a chauffeur. Hearing of left ear still impaired. Reflexes active but otherwise negative. Fundi negative. No impairment of speech apparently, except for "catch" and test phrases, such as "truly rural"; "around the rugged rock the ragged rascal ran"; "the third red riding artillery brigade," etc., when the patient slurs the words occasionally.

Examination (September 20, 1915—30 months after injury).—No complaints; patient, however, is becoming alcoholic and at this examination is slightly intoxicated. Hearing of left ear has improved and bone conduction is no longer greater than air conduction. Reflexes active but otherwise negative. Fundi negative. Speech was not tested at this examination on account of the patient's emotional instability due to alcohol. Decompression areas depressed and pulsating normally.

Examination (September 20, 1917—53 months after injury).—Patient has just returned from France, where he has been driving an ambulance during the past year. No complaints and "equal to three Germans." Alcoholic no longer. No impairment of hearing of left ear can be elicited. Slight impairment of speech can still be ascertained by test phrases only. Hearing negative. Reflexes active but otherwise negative. Fundi negative.

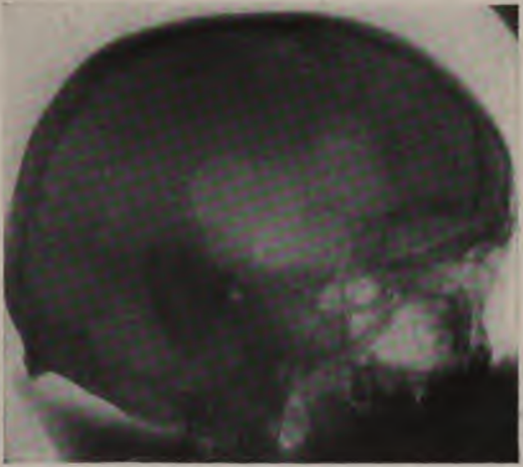

FIG. 73.—Lateral röntgenogram showing the bony defects of the bilateral decompression operation to lower an extreme intracranial pressure due to subdural and intracerebral hemorrhages. Excellent recovery. Note the prominent external occipital protuberance.

Last Examination (September 28, 1918—65 months after injury).—Patient writes from France that he is "as well as ever."

Remarks.—In these patients having a high intracranial pressure and when one decompression does not lessen the intracranial pressure markedly so that the brain can pulsate normally, then a second decompression should be performed immediately if the general condition of the patient warrants it; if the general condition of the patient, however, is such that an immediate second operation would be too dangerous, then it would be advisable to wait for a period of from 24 to 48 hours before attempting it. These bilateral decompressions are usually necessary if the brain is "water-logged" and yet "dry"—that is, a swollen brain but very little cerebrospinal fluid escapes at the time of the first operation and therefore rendering a second operation necessary. The recovery of speech in a patient whose motor speech area was accurately observed at operation to be lacerated and through which large blood-clots were extruded, tends to confirm the belief as formed from similar lesions in other patients, that the impairment of speech in these patients must be due to a local compression of the cortical cells due to blood-clot and edema rather than one of actual destruction of the cells of motor speech—otherwise there would be no return of function.

This patient has been examined by me repeatedly since the operation and he is apparently normal mentally, emotionally and physically. It is a remarkable case—not only on account of the recovery of his life but the complete return of his former normal condition; at the time of the operation, when large dark clots were seen welling up through the substance of the brain, it was not conceived possible that this patient could return to his former normality. This case illustrates the advisability of performing a bilateral operation when it seems improbable that the one decompression will be sufficient to lessen the increased intracranial pressure entirely; it is not so much a question of hemorrhage or edema (that is, an excess amount of cerebrospinal fluid), but rather a question of the amount of increased intracranial pressure; whether it be due to hemorrhage or edema, it matters not—the chief object of the treatment being to lessen the pressure.

It is interesting to note that frequently the laceration of the brain and the intracranial hemorrhage may be found in one hemisphere, and yet all of the signs obtained at the examinations point to the other cerebral hemisphere as being the one greater damaged; it is difficult to explain this apparent paradox—unless due to *contre-coup* or to a subcortical hemorrhage, which is not ascertained when the bilateral decompression is performed; as the condition, however, frequently improves so quickly following operation, this latter explanation is only probable. It is possible that the greater compression being over one hemisphere, in some manner forces the other hemisphere against the overlying vault of the skull and thus produces the clinical signs of a greater impairment of the other hemisphere. The intracranial lesions in patients of this character are so multiple that it is most difficult to explain satisfactorily the causes of all of the clinical signs merely from the observations at the two operations which reveal only comparatively small portions of the entire brain itself. When the clinical signs are very confusing in these patients, it is better surgical judgment to perform a right subtemporal decompression first and then the left subtemporal decompression would be easier technically and of less danger to the underlying cerebral cortex.

Case 49.—Acute severe brain injury; signs of extreme intracranial pressure; subdural and intracerebral hemorrhage. Bilateral decompression and drainage. Recovery.

No. 079.—Andrew. Twenty-five years. White. Single. Clerk. U. S.

Admitted November 30, 1913, Muhlenburg Hospital, Plainfield, N. J. Referred by Doctor B. Van D. Hedges.

Operations—December 3 and 16, 1913—6 and 22 days, respectively, after injury. Bilateral decompression and drainage.

Discharged February 2, 1914—43 days after second operation.

Family history negative.

Personal history negative.

Present Illness.—Three days ago, while patient was riding a motorcycle, he collided with a stone wall; immediate loss of consciousness; brought to the hospital immediately in profound shock, unconscious and has remained unconscious; profuse bleeding from left ear.

Examination (3 days after admission).—In consultation with Doctor Hedges: temperature, 100.6°; pulse, 78; respiration, 20; blood-pressure, 144. Well developed and nourished. Unconscious and cannot be aroused. Incontinence of urine and feces. Both orbits ecchymotic and bilateral subconjunctival hemorrhages. Dry blood in left auditory canal; left mastoid ecchymosis. Marked edema and apparent tenderness over left squamous region. Pupils equal and react to light normally. Reflexes: patellar exaggerated, especially right; bilateral Babinski and ankle clonus; abdominal reflexes present and equal. Fundi: retinal veins dilated; edematous blurring of entire nasal halves of both optic disks. Lumbar puncture—cerebrospinal fluid bloody and under very high pressure (approximately 20 mm.).

Treatment.—An immediate left subtemporal decompression advised to lessen the high intracranial pressure.

First Operation (3 days after admission).—Left subtemporal decompression: usual vertical incision, bone removed and no complications; much free blood and ecchymosis of temporal muscle beneath the temporal fascia and thus indicating a fracture of the underlying bone; this observation was confirmed upon retracting the fibres of the temporal muscles and exposing a horizontal linear fracture of the squamous portion of left temporal bone, extending forward and backward beyond the margins of the decompression opening. Small extradural clot evacuated; dura very tense and, upon incising it, bloody cerebrospinal fluid spurted to a height of 8–10 inches for a period of one minute. Upon enlarging dural opening, the "wet" edematous cortex tended to protrude under high pressure, owing to the rapid escape of much cerebrospinal fluid and blood; the "sweating" of the arachnoid was very marked, so that the brain became less tense before the end of the operation. Just below the Sylvian fissure, dark-clotted blood of the consistency of currant jelly welled out through a cortical laceration of 2 cm. in length and small blood-clots continued to extrude during the operation. Numerous punctate hemorrhages throughout the cortex. Usual closure with 2 drains of rubber tissue inserted. Duration, 1 hour. Post-operative notes: decompression area remained unusually tense, and as the patient did not become entirely conscious but remained delirious and stuporous, I examined the patient for the second time on December 16, 1913—13 days after the first operation: the decompression area was very tense—so much so that it could not pulsate; the left reflexes were greater than the right, a left Babinski and left ankle clonus were present, while the fundi showed a blurring of the nasal halves of the optic disks and a dilatation of the retinal veins; therefore, as the intracranial pressure was still very high, it was thought advisable to perform a similar decompression and drainage upon the other side of the head.

Second Operation (December 16, 1913—13 days after the first operation).—Right subtemporal decompression: usual vertical incision, bone removed and no complications; temporal muscle beneath the temporal fascia ecchymosed and beneath it in the squamous portion of the temporal bone were 2 irregular fractures extending downward into the base. Dura very tense and bluish; upon incising it, blood-tinged cerebrospinal fluid spurted under high pressure, and upon enlarging dural opening a very "wet" edematous cortex with many punctate hemorrhages was exposed; it tended to protrude but did not rupture, and at the end of the operation the brain pulsated normally, owing to the loss of much cerebrospinal fluid. Usual closure with 2 drains of rubber tissue inserted. Duration, 45 minutes.

Post-operative Notes.—Uneventful operative recovery: patient began to improve immediately after the operation; became conscious upon the second day, and although he remained mentally confused for almost a week, yet that condition gradually lessened so that the operative recovery was excellent; the Babinski reflex could not be obtained for the first time on the 16th day after operation.

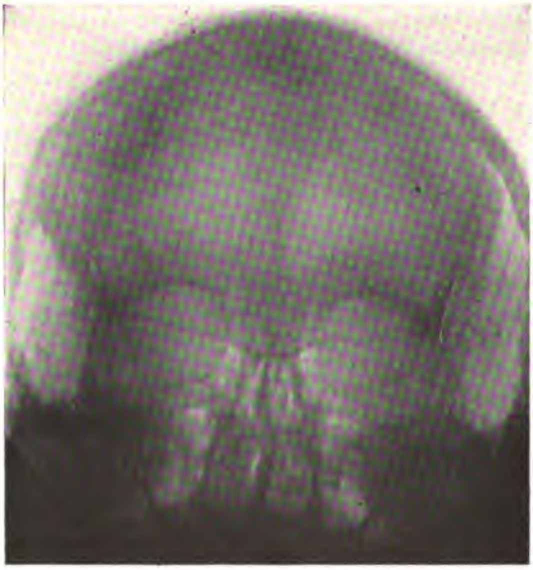

FIG. 74.—Bony defects of bilateral decompression operation clearly shown; note the greater removal of bone of the left decompression. The high intracranial pressure due to subdural and intracerebral hemorrhages successfully lowered. Excellent recovery.

Examination at discharge (62 days after admission).—Temperature, 98.8°; pulse, 70; respiration, 18; blood-pressure, 136. Patient feels well; occasional headache when stooping; rather difficult for patient to think consecutively. "Can't concentrate my thoughts"; perseveration of words at times. Definite impairment of hearing of both ears—bone conduction being greater than air conduction; otoscopic examination reveals a healing laceration of the posterior portion of left tympanic membrane, whereas the right tympanic membrane is intact, but thickened and retracted. Patient becomes fatigued after any exertion. Decompression areas slightly depressed; normal pulsation. Pupils equal and react normally. Reflexes very active but otherwise negative. Fundi: retinal veins of normal size; no edema of optic disks but along the nasal margins and in the physiological cups there is a small amount of new tissue formation—that is, a very mild degree of secondary optic atrophy due to the former high intracranial pressure being prolonged over a period of weeks.

Examination (June 7, 1914—7 months after injury).—No serious complaints; patient is at work daily in an office where it is not strenuous. Talks rather slowly, with a definite retardation of mentality. Easily fatigued and always before the end of the day. Both decompression areas depressed; normal pulsation. Hearing of left ear impaired; bone conduction is greater than air conduction. Reflexes active but otherwise negative. Fundi: no edema of margins of optic disks but a slight amount of new tissue formation along the nasal margins; retinal veins of normal size. X-ray (Doctor A. J.

Quimby)—"the areas of bony decompression revealed; no lines of fracture observed" (Fig. 74).

Examination (June 10, 1916—31 months after injury).—There is still a definite retardation mentally and a marked slowness of speech; easily fatigued so that patient is unable to do hard physical work for a number of hours. Patient, however, works daily and is able to support himself. Hearing of left ear still impaired; right ear almost normal. Reflexes active but otherwise negative. Fundi, the same as at preceding examination.

Examination (May 10, 1918—54 months after injury).—Patient has just returned from an army cantonment where he has been chauffeur; he was able to perform this work satisfactorily, but succeeded in obtaining his discharge in order to enlist in the regular army and desires a certificate of good health. No complaints. "I feel better and stronger now than at any time since the injury." Both decompression areas depressed; difficult to elicit pulsation by palpation. Hearing of left ear still impaired; bone conduction is greater than air conduction. Reflexes active but otherwise negative. Fundi: retinal veins of normal size; nasal margins of both optic disks irregular from new tissue formation. Limited service in the army was considered advisable. A photograph was taken May 8, 1918 (Fig. 75).

FIG. 75.—This patient had an extreme intracranial pressure due to subdural and intracerebral hemorrhages but no fracture of the skull; successfully treated by a bilateral subtemporal decompression and drainage.

Remarks.—It is to be regretted that the bilateral decompression was not performed at the time of the first operation—that is, 3 days after the injury; it was not advised at that time, however, as it was thought that the one decompression would be sufficient. This period of high, increased pressure persisting almost 3 weeks was sufficient to impair the cortical cells functionally, at least for a period of years, and possibly permanently. This case illustrates the necessity of an early lessening of the increased intracranial pressure as soon as possible after the initial shock of the head injury has disappeared—not only as a means of possibly saving the life of the individual, but, very important, to obtain as normal an individual as before the injury, or at least to approximate the former normality.

The permanent impairment of hearing of the left ear and referable to the middle ear, is undoubtedly due to scar tissue formation and retraction of the left tympanic membrane and possibly some impairment of the ossicular transmitting mechanism of the left middle ear. As a rule, however, the impairment of hearing in these patients referable to the middle ear is of temporary duration only—12 to 18 months,—whereas if the condition has been complicated by an otitis media or by an unusual amount of new tissue greatly lessened by the drainage of this small amount of fluid. Cortex at end of operation, however, did pulsate but very slightly. Usual closure with 2 drains of rubber tissue inserted. Duration, 80 minutes.

Post-operative Notes.—Patient became conscious 6 hours after operation, although he was very much mentally confused and mildly delirious. Twenty-four hours later, patient sat up in bed saying "I want to go home, take this bandage off"; the pulse was 64. Five days after operation, in an attempt to remove the gauze packing used to control the hemorrhage at the base during the operation, the bleeding became very profuse, requiring another gauze tape packing to be used. On the tenth day after operation, the sutures were all removed and also the gauze packing. Patient has been mildly delirious; it was now ascertained that the patient has been a drug addict and upon his receiving the appropriate treatment by Doctor E. S. Bishop for the drug addiction the patient began to improve immediately. Incisions healed *per primam*.

Examination at discharge (21 days after admission).—(Patient transferred to Bellevue Hospital at the request of Doctor E. S. Bishop so that he could be properly controlled for his drug addiction.) Temperature, 99.6°; pulse, 68; respiration, 20; blood-pressure, 144. Both decompression areas bulge slightly beyond the flush of scalp; normal pulsation. Pupils contracted equally (morphia). Reflexes: patellar very active—left being possibly greater than right; suggestive left Babinski; abdominal reflexes present and equal. Fundi—retinal veins enlarged; edematous blurring of nasal margins of both optic disks, but other details of disks clear and distinct.

Report (September 20, 1916—31 months after injury).—Patient was met while at work in another hospital; is able to do his work, but it is believed that he is still a drug addict. Decompression openings did not bulge and patient had no complaints referable to the former head injury.

Last Report (June 16, 1918—52 months after injury).—Patient works daily as an orderly in the hospital; no complaints referable to the former head injury. Both decompression areas are depressed. Patient admits his drug addiction but refuses treatment.

Remarks.—It is hardly conceivable that a patient having such a severe brain injury as this patient could recover so well; especially so, when it is remembered that not only was the brain lacerated but that throughout the exposed cortex there were so many punctate hemorrhages that it had almost the appearance of liver tissue. It merely tends to confirm the opinion, however, that the danger of brain injuries is not so much due to the laceration or hemorrhage in the brain substance as it is the effect of high intracranial pressure being permitted to remain during a period of days and weeks—not only so far as life itself is concerned, but as to the future normality of the patient. It must be remembered also that the part of the brain exposed by the decompression operation is merely the temporosphenoidal lobe, the lower portion of the parietal lobe and at times the posterior portion of the contiguous frontal lobe, and that these portions of the cortex are, in the right cerebral hemisphere in right-handed individuals, comparatively "silent" areas, and even in the left cerebral hemi-

sphere in right-handed patients, if we exclude the motor speech area in the posterior portion of the third left frontal convolution; any damage to the cortex immediately beneath the decompression opening is only ascertained clinically with difficulty and by special tests, so that the large area of the cerebral cortex, not exposed by the decompression operations, may not be in some of these patients injured at all, and yet the area of the cortex exposed at operation may be lacerated and exceedingly hemorrhagic; frequently at autopsies upon patients having had brain injuries and yet no operation having been performed, the areas of the cortex most frequently lacerated and contused are the temporo-sphenoidal lobes, then the frontal lobes and finally the occipital lobes, and in this order of frequency.

It would have been better judgment surgically if, upon ascertaining that the intradural pressure was so extremely high, a lumbar puncture had been made and cerebrospinal fluid withdrawn just before the dura at the first operation was opened; in this manner the intradural pressure would have lessened and there would have been less risk of the underlying cerebral cortex rupturing; a ventricle puncture might also have been attempted before opening the dura widely. Although no clinical signs of this cortical rupture persisted, yet it certainly does the brain and the patient no good, and this complication technically, whenever possible, should be avoided.

The small contracted pupils of the patient as observed upon admission to the hospital were, at that time, considered as being due to severe cortical irritation; the factor of drug-addiction disclosed later may have been the cause of these pin-point pupils (as the condition persisted even after the discharge of the patient from the hospital), and yet this patient did have sufficient cortical irritation to produce this extreme miosis.

ACUTE SEVERE BRAIN INJURIES ASSOCIATED WITH A HIGH INTRACRANIAL PRESSURE DUE TO CEREBRAL EDEMA ALONE AND REQUIRING THE CRANIAL OPERATION OF SUBTEMPORAL DECOMPRESSION AND DRAINAGE.

It is this factor of acute cerebral edema (an increased amount of cerebrospinal fluid), which results in varying degree from brain injuries and in fact from so many cranial injuries alone, with and without a fracture of the skull, that has been so frequently overlooked in the past, and it is this complication of cerebral edema which makes these injuries such serious ones, both in their immediate and remote effects. It is not definitely known whether this excess of cerebrospinal fluid is due to its increased secretion or to its diminished absorption and excretion, although the latter cause is more probably the correct one.

Even in cranial injuries not more severe than to produce a cerebral "concussion"—in many of these patients there is demonstrated a mild increase of the pressure of the cerebrospinal fluid, as elicited by careful ophthalmoscopic examinations and by the spinal mercurial manometer; this latter test reveals the cerebrospinal fluid clear and of normal cell count, whereas the pressure may be increased to 11–12 mm (normal 5–9 mm.); these patien[illegible] continue to have headache and the other symptoms and signs of a mild [illegible] the intracranial pressure, until the expectant palliative treatm[illegible]es the gradual absorption of the excess cerebrospinal fluid

sphere in right-handed patients, if we exclude the motor speech area in the posterior portion of the third left frontal convolution; any damage to the cortex immediately beneath the decompression opening is only ascertained clinically with difficulty and by special tests, so that the large area of the cerebral cortex, not exposed by the decompression operations, may not be in some of these patients injured at all, and yet the area of the cortex exposed at operation may be lacerated and exceedingly hemorrhagic; frequently at autopsies upon patients having had brain injuries and yet no operation having been performed, the areas of the cortex most frequently lacerated and contused are the temporo-sphenoidal lobes, then the frontal lobes and finally the occipital lobes, and in this order of frequency.

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ACUTE SEVERE BRAIN INJURIES ASSOCIATED WITH A HIGH INTRACRANIAL PRESSURE DUE TO CEREBRAL EDEMA ALONE AND REQUIRING THE CRANIAL OPERATION OF SUBTEMPORAL DECOMPRESSION AND DRAINAGE.

It is this factor of acute cerebral edema (an increased amount of cerebrospinal fluid), which results in varying degree from brain injuries and in fact from so many cranial injuries alone, with and without a fracture of the skull, that has been so frequently overlooked in the past, and it is this complication of cerebral edema which makes these injuries such serious ones, both in their immediate and remote effects. It is not definitely known whether this excess of cerebrospinal fluid is due to its increased secretion or to its diminished absorption and excretion, although the latter cause is more probably the correct one.

Even in cranial injuries not more severe than to produce a cerebral "concussion"—in many of these patients there is demonstrated a mild crease of the pressure of the cerebrospinal fluid, as elicited by careful hthalmoscopic examinations and by the spinal mercurial manometer; this tter test reveals the cerebrospinal fluid clear and of normal cell count, ereas the pressure may be increased to 11–12 mm (normal 5–9 mm.); these tients continue to have headache and the other symptoms and signs of a ild increase of the intracranial pressure, until the expectant palliative eatment facilitates the gradual absorption of the excess cerebrospinal fluid formation in the middle ear and if the laceration of the tympanic membrane should not be repaired perfectly, then there is always a more or less degree of deafness resulting.

If it had been realized in this patient that the increased intracranial pressure was so extreme, then undoubtedly the first operation would have been performed upon the right side of the head for fear of a possible injury to the motor speech area if a left decompression should be performed; however, as the clinical signs of this patient indicated a greater lesion of the left cerebral hemisphere and since the intracranial pressure could not then be so accurately estimated as it can be now, the left subtemporal decompression and drainage was performed first, and fortunately no operative damage occurred; it was unfortunate, however, that an immediate bilateral decompression was not performed, as it would be now without any hesitation under similar conditions of extreme intracranial pressure. These patients having such extreme intracranial pressure are rare, but when they do occur then the appropriate bilateral decompression and drainage should be performed; it seems that strong, well-nourished and well-developed adults between 20 and 35 years of age who have greater powers of resistance—these are the patients having cranial injuries who frequently develop the highest intracranial pressure, and this is naturally explained upon physiological grounds.

The indefinite emotional instability of this patient and the vague mental retardation as exhibited at a number of the examinations during the past 5 years are due undoubtedly to this extreme intracranial pressure persisting acutely for over 3 weeks following the injury, and also to the definite damage of the cerebral cortex itself from the punctate hemorrhages, lacerations and supracortical hemorrhages. This patient, in my opinion, would have died unless the cranial operations had been performed, and it is to be regretted that they were not performed as early as possible following the injury.

Case 50.—Acute severe brain injury; signs of extreme intracranial pressure associated with extradural, subdural, cortical and intracranial hemorrhage. Bilateral decompression and drainage. Recovery.

No. 109.—John. Twenty-seven years. White. Single. Orderly. U. S.

Admitted February 25, 1914, Polyclinic Hospital. Referred by Doctor John A. Bodine.

Operation February 25, 1914—2½ hours after injury. Bilateral decompression and drainage.

Discharged March 16, 1914—20 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While intoxicated, patient fell from a taxicab; immediate loss of consciousness; brought to the hospital in a taxicab.

Examination upon admission (20 minutes after injury).—Temperature, 99°; pulse, 66; respiration, 16; blood-pressure, 140. Semiconscious upon admission—could be easily aroused and talked incoherently and irrelevantly; within 20 minutes, however, and during the examination, it became increasingly difficult to arouse patient so that he became profoundly unconscious. Contusion over top of head. Profuse bleeding from the nose, but none from mouth or ears. Pupils very small and contracted but equal. Reflexes: patellar—left greater than right; suggestive left Babinski; abdominal reflexes cannot be obtained. Fundi—retinal veins dilated; nasal margins of both optic disks blurred by edema. Lumbar puncture—bloody cerebrospinal fluid under high pressure (approximately 20 mm.).

Treatment.—During the examination and within 1 hour later, patient became profoundly unconscious, pulse descended to 60, respiration to 14 and of the Cheyne-Stokes type of irregularity; a definite left Babinski was elicited and the ophthalmoscope revealed an obscuration of the nasal halves and also the temporal margins of both optic disks. As the intracranial pressure was undoubtedly increasing and very rapidly, an immediate right subtemporal decompression was considered advisable. While waiting for the operating room to be prepared, Doctor A. J. Quimby made a röntgenogram which disclosed a "linear fracture of the frontal bone" (Fig. 76).

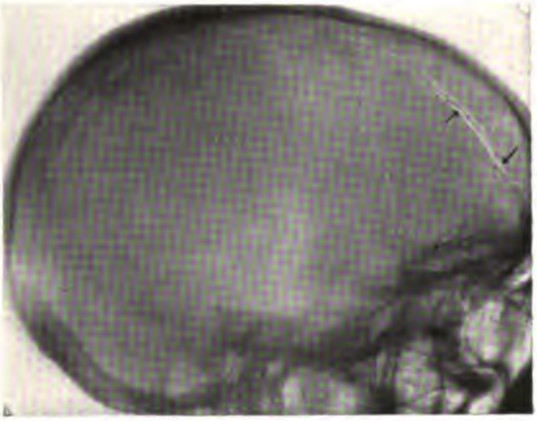

FIG. 76.—Oblique linear fracture of right frontal bone in a patient having a very high intracranial pressure due to extradural, subdural and intracerebral hemorrhages. Recovery following a bilateral subtemporal decompression.

First Operation (2 hours after admission).—Right subtemporal decompression: usual vertical incision, removal of bone and no complications. Large extradural clot covered the lower half of the operative field and dark clots—the size of English walnuts—welled up from the base extradurally. Upon evacuating this extradural hemorrhage, the dura which had been depressed was very tense and of a dark blue tint, and upon incising it, large subdural clots welled up from the base. The intradural tension was so high that the underlying cortex, which was filled with punctate hemorrhages, ruptured transversely to a length of 3 inches and even 2 inches in width; much dark clotted blood extruded from this laceration and the entire cortex tended to protrude through the bony opening—gauze tape packing being necessary to control the hemorrhage coming from the base. Owing to this high cerebral pressure, which apparently could not be entirely relieved by the unilateral decompression, it was considered advisable to perform a similar decompression upon the opposite side of the head. Usual closure with 2 drains of rubber tissue inserted. Temporary sterile gauze dressing applied.

Second Operation.—Left subtemporal decompression: usual vertical incision, bone removed and no complications. No extradural clot. Dura very tense and upon incising it, the hemorrhagic cortex—almost the appearance of liver tissue—tended to protrude but did not rupture; a small amount [illegible]od-tinged cerebrospinal fluid oozed through the dural opening but the [illegible]vas so swollen and "water-logged" that the pressure was not greatly lessened by the drainage of this small amount of fluid. Cortex at end of operation, however, did pulsate but very slightly. Usual closure with 2 drains of rubber tissue inserted. Duration, 80 minutes.

Post-operative Notes.—Patient became conscious 6 hours after operation, although he was very much mentally confused and mildly delirious. Twenty-four hours later, patient sat up in bed saying "I want to go home, take this bandage off"; the pulse was 64. Five days after operation, in an attempt to remove the gauze packing used to control the hemorrhage at the base during the operation, the bleeding became very profuse, requiring another gauze tape packing to be used. On the tenth day after operation, the sutures were all removed and also the gauze packing. Patient has been mildly delirious; it was now ascertained that the patient has been a drug addict and upon his receiving the appropriate treatment by Doctor E. S. Bishop for the drug addiction the patient began to improve immediately. Incisions healed *per primam.*

Examination at discharge (21 days after admission).—(Patient transferred to Bellevue Hospital at the request of Doctor E. S. Bishop so that he could be properly controlled for his drug addiction.) Temperature, 99.6°; pulse, 68; respiration, 20; blood-pressure, 144. Both decompression areas bulge slightly beyond the flush of scalp; normal pulsation. Pupils contracted equally (morphia). Reflexes: patellar very active—left being possibly greater than right; suggestive left Babinski; abdominal reflexes present and equal. Fundi—retinal veins enlarged; edematous blurring of nasal margins of both optic disks, but other details of disks clear and distinct.

Report (September 20, 1916—31 months after injury).—Patient was met while at work in another hospital; is able to do his work, but it is believed that he is still a drug addict. Decompression openings did not bulge and patient had no complaints referable to the former head injury.

Last Report (June 16, 1918—52 months after injury).—Patient works daily as an orderly in the hospital; no complaints referable to the former head injury. Both decompression areas are depressed. Patient admits his drug addiction but refuses treatment.

Remarks.—It is hardly conceivable that a patient having such a severe brain injury as this patient could recover so well; especially so, when it is remembered that not only was the brain lacerated but that throughout the exposed cortex there were so many punctate hemorrhages that it had almost the appearance of liver tissue. It merely tends to confirm the opinion, however, that the danger of brain injuries is not so much due to the laceration or hemorrhage in the brain substance as it is the effect of high intracranial pressure being permitted to remain during a period of days and weeks—not only so far as life itself is concerned, but as to the f normality of the patient. It must be remembered also that the part brain exposed by the decompression operation is merely the te sphenoidal lobe, the lower portion of the parietal lobe and at ti posterior portion of the contiguous frontal lobe, and that these of the cortex are, in the right cerebral hemisphere in right-handed uals, comparatively "silent" areas, and even in the left cerebra

sphere in right-handed patients, if we exclude the motor speech area in the posterior portion of the third left frontal convolution; any damage to the cortex immediately beneath the decompression opening is only ascertained clinically with difficulty and by special tests, so that the large area of the cerebral cortex, not exposed by the decompression operations, may not be in some of these patients injured at all, and yet the area of the cortex exposed at operation may be lacerated and exceedingly hemorrhagic; frequently at autopsies upon patients having had brain injuries and yet no operation having been performed, the areas of the cortex most frequently lacerated and contused are the temporo-sphenoidal lobes, then the frontal lobes and finally the occipital lobes, and in this order of frequency.

It would have been better judgment surgically if, upon ascertaining that the intradural pressure was so extremely high, a lumbar puncture had been made and cerebrospinal fluid withdrawn just before the dura at the first operation was opened; in this manner the intradural pressure would have lessened and there would have been less risk of the underlying cerebral cortex rupturing; a ventricle puncture might also have been attempted before opening the dura widely. Although no clinical signs of this cortical rupture persisted, yet it certainly does the brain and the patient no good, and this complication technically, whenever possible, should be avoided.

The small contracted pupils of the patient as observed upon admission to the hospital were, at that time, considered as being due to severe cortical irritation; the factor of drug-addiction disclosed later may have been the cause of these pin-point pupils (as the condition persisted even after the discharge of the patient from the hospital), and yet this patient did have sufficient cortical irritation to produce this extreme miosis.

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It is this factor of acute cerebral edema (an increased amount of cerebrospinal fluid), which results in varying degree from brain injuries and in fact from so many cranial injuries alone, with and without a fracture of the skull, that has been so frequently overlooked in the past, and it is this complication of cerebral edema which makes these injuries such serious ones, both in their immediate and remote effects. It is not definitely known whether this excess of cerebrospinal fluid is due to its increased secretion or to its diminished absorption and excretion, although the latter cause is more probably the correct one.

Even in cranial injuries not more severe than to produce a cerebral "concussion"—in many of these patients there is demonstrated a mild increase of the pressure of the cerebrospinal fluid, as elicited by careful ophthalmoscopic examinations and by the spinal mercurial manometer; this latter test reveals the cerebrospinal fluid clear and of normal cell count, whereas the pressure may be increased to 11–12 mm (normal 5–9 mm.); these patients continue to have headache and the other symptoms and signs of a mild increase of the intracranial pressure, until the expectant palliative treatment facilitates the gradual absorption of the excess cerebrospinal fluid and then the headaches, etc., cease and there is no longer an increased intracranial pressure, as revealed by the ophthalmoscope and the spinal manometric tests—and this is the usual result. Chronic alcoholism and chronic renal diseases as well as any chronic toxemic condition permit and apparently predispose these patients to the formation of cerebral edema, and the prognosis in these patients is always more grave.

An acute cerebral edema, however, following a brain injury, with and without a fracture of the skull, may produce such an increase of the intracranial pressure that the danger of medullary compression is an imminent one, and if extreme or prolonged then the great risk of the onset of acute medullary edema—and the death of the patient. The autopsy records of these patients disclose such a "wet" edematous condition of the brain and of the medulla itself that medullary compression and its end-result, medullary edema, had resulted; a fracture of the skull may or may not be present, and in some patients the cerebrospinal fluid may be blood-tinged, although the amount of blood was so small that it did not increase the intracranial pressure to any appreciable degree. It is in this type of patient in whom no intracranial hemorrhage nor even a fracture of the skull was demonstrated and yet having a high intracranial pressure—these are the patients who rarely received the appropriate treatment of an early cranial decompression and drainage, and it is in this group of patients having cranial injuries that the greatest advance in treatment has occurred in recent years and a larger percentage of recoveries can still be obtained; the pathology of the condition is now recognized so that the treatment will thus be an earlier and more rational one.

Recent severe brain injuries, with and without a fracture of the skull, and associated with high intracranial pressure due, not to large intracranial hemorrhage, but to the so-called cerebral edema (an increased amount of cerebrospinal fluid). Subtemporal decompression and drainage. Recovery.

CASE 51.—Acute severe brain injury; signs of high intracranial pressure due to cerebral edema. Left subtemporal decompression and drainage. Excellent recovery.

No. 025. Patrick. Thirty-one years. White. Single. Stevedore. Ireland.

Admitted May 8, 1913, Polyclinic Hospital. Referred by Doctor A. B. Morrow.

Operation May 8, 1913—18 hours after injury. Left subtemporal decompression and drainage.

Discharged May 16, 1913—8 days after operation.

Family history negative.

Personal history negative, except for chronic alcoholism.

Present Illness.—Sixteen hours ago, while in a bar-room fight, patient was repeatedly struck over the head by some heavy, blunt object; was unconscious for several minutes, and when the police arrived patient was in a stuporous, drowsy condition. Taken to the station house and registered as "intoxicated"; during the next 12 hours, patient vomited repeatedly and bled profusely from the left ear; as bleeding from the left ear lessened, patient gradually became more and more stuporous and finally completely unconscious; brought to the hospital in the patrol wagon.

Examination upon admission (16 hours after injury).—Temperature, 99.8°; pulse, 60; respiration, 16; blood-pressure, 142. Semiconscious but lapses into total unconsciousness; in the intervals, he can be aroused by firm supra-orbital pressure. Multiple lacerations of scalp; over left side of head are several very tender areas. Left orbit very ecchymotic. Clotted blood in left auditory canal; otoscopic examination reveals a tear of the posterior portion of the left tympanic membrane; left mastoid ecchymosis. Pupils—left contracted and does not react to light. Reflexes—patellar very active and equal; no ankle clonus but suggestive right Babinski; abdominal reflexes absent. Fundi—retinal veins dilated; obscuration of entire nasal halves and temporal margins of both optic disks—left possibly more than right. Lumbar puncture—clear cerebrospinal fluid under high pressure (approximately 18 mm.); laboratory report (Doctor W. A. MacFarlane)—"an occasional red blood-cell in the cerebrospinal fluid."

Treatment.—An immediate left subtemporal decompression and drainage advised.

Operation (18 hours after injury and 2 hours after admission).—Left subtemporal decompression: usual vertical incision, bone removed and no complications. Dura very tense and upon incising it, clear cerebrospinal fluid spurted to a height of almost 3 inches; upon enlarging the dural opening much clear cerebrospinal fluid escaped under pressure, exposing a very "wet" edematous cortex which tended to protrude but did not rupture; cortex receded and pulsated at the end of the operation. No cortical hemorrhage nor laceration observed—merely a "wet" edematous brain. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Excellent operative recovery; patient became conscious within 12 hours after the operation and remained clear mentally; all sutures removed upon the fifth day—incision healing *per primam.*

Examination at discharge (8 days after operation and 9 days after injury; patient was taken to court to answer charges of assault and was sentenced to jail for 3 months).—Temperature, 99°; pulse, 72; respiration, 18; blood-pressure, 136. No complaints other than "heavy feeling" in the head; "light-headed," especially in the morning. Operative wound has healed and bulges slightly beyond the flush of scalp; pulsates normally. Hearing of left ear impaired; bone conduction greater than air conduction. Pupils equal and react normally. Reflexes: patellar active but equal; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi—retinal veins enlarged; nasal margins of both optic disks rather hazy but otherwise negative.

Treatment.—General hygienic measures urged, and especially the avoidance of alcohol.

Examination (September 20, 1916—40 months after injury).—(Patient has been brought to hospital following another fight in which he has received a fractured clavicle.) Except for the general effects of chronic alcoholism, patient is in good condition. Decompression area depressed; owing to new bone formation, no pulsation is palpable. Hearing of left ear less acute; bone conduction equals air conduction. Reflexes sluggish but otherwise negative. Fundi—general retinal congestion and suffusion but optic disks clear and distinct.

Last Report (June 17, 1918—60 months after injury).—Letter from patient states that he is in Sing Sing on a third degree charge of homicide while intoxicated; sentenced to 5 years' imprisonment. No complaints referable to head injury. Hopes by good behavior to obtain an earlier release and then "start over again."

Remarks.—It is interesting to record the observation made by the policeman in charge of this patient in the station house: "The pulse which had been around 80 while the ear was bleeding became within 2 hours 66, and even 64 after the bleeding from the ear had lessened" (this officer had studied medicine before entering the service). The explanation of this observation is simple: as long as the intracranial hemorrhage in the cerebrospinal fluid was escaping through the fracture and into the external auditory canal, just so much was the intracranial pressure lessened, and thus the signs of an increased pressure did not occur; but when the escape of cerebrospinal fluid was blocked in the ear itself, then this accumulation of excess cerebrospinal fluid increased the intracranial pressure until the effect of it was to be seen in the fundi, in the measurement of the pressure of the cerebrospinal fluid at lumbar puncture and by the signs of mild medullary compression as exhibited by the lowering of the pulse- and respiration-rate. Undoubtedly in many patients, this escape of blood and cerebrospinal fluid through a cranial fracture into the ears or nose is sufficient to prevent a high increase of the intracranial pressure and thus a sort of "natural" decompression is performed and frequently a decompression operation is thus avoided, although at the risk of infection and a possible meningitis through the fracture into the ear, and particularly into the nose and pharynx.

The laboratory report of the cerebrospinal fluid indicating the presence of a very small amount of blood, might be due either to the intracranial condition, or, and much more probable in view of the operative findings, to the technic of the lumbar puncture itself; it is very difficult at times not to contaminate cerebrospinal fluid by a small amount of free blood due to the lumbar puncture itself.

The gradual improvement of the hearing so that at the examination 40 months after injury, the bone conduction equalled the air conduction, would not indicate that the impairment of hearing of the left ear would eventually be normal; those patients in whom the impairment of hearing becomes normal, do so within 12 or 18 months following the injury, and the longer it takes for the improvement of hearing to occur, the greater is the amount of scar tissue and retraction of the tympanic membrane and ossicles so that a complete recovery of hearing becomes more and more doubtful.

The absence of intracranial hemorrhage in this patient does not mean that the patient was not seriously injured and in danger of his life, because it is not the intracranial hemorrhage that is the serious factor in these cases of brain injuries, but rather the degree of increased intracranial pressure; to a certain extent, it is not really a brain injury but the pressure effect, if continued, will produce both immediate and remote damage to the cerebral cortex, and of greater danger as far as life is concerned, to the medulla itself.

It is a common observation in these patients who have been at all alc that, following a head injury, they are much more liable to an acute ce edema than are the patients who are not alcoholic; and also patients tomed to the daily use of alcohol are much more liable to be upset, bot tally and emotionally, not only more severely but for a much longer pe time. These patients, therefore, should not be allowed to return to former active lives for a period of at least 6 months, and not then they have returned to their former normal stability; they should be st urged not to use alcohol in any form as it renders these patients muc liable to future complications, such as epileptiform spells and pro emotional impairments and states of mental confusion, even to the of traumatic dementia. These patients also are comparatively bad ope risks and this factor of alcoholism must always be considered when question of an operation or not.

In many of these patients, it is better surgical judgment to perf bilateral subtemporal decompression if the intradural pressure is ext high and whenever later it is ascertained that the decompressio bulges tensely for a period of at least 10 days; the fact, however, th cortex pulsates practically normally after the escape of a large amo cerebrospinal fluid at the time of the operation, indicates, as a rule, bilateral decompression will not be necessary. The rather delaye valescence with emotional instability may probably have been due t prolonged increase of intracranial pressure associated with the fac alcoholism, and not to a primary injury to the brain itself.

CASE 52.—Acute severe brain injury; signs of high intracranial pr due to cerebral edema. Right subtemporal decompression and dra Excellent recovery.

No. 030.—Vincent. Twenty-eight years. White. Single. Mec United States.

Admitted June 2, 1913, Polyclinic Hospital. Referred by J. A. Bodine.

Operation June 2, 1913—6½ hours after injury. Right subten decompression and drainage.

Discharged June 15, 1913—13 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While working in a new building, patient fell a di of 2 stories upon a wooden floor; only momentary loss of consciousne was able to walk to the ambulance which brought him to the hospita

Examination upon admission (35 minutes after injury).—Ten ture, 99.2°; pulse, 64; respiration, 16; blood-pressure, 144. Con Patient says he "feels all right," except for a severe headache and a f of "giddiness." Well-developed and nourished; no alcoholism. Has ited twice without nausea. Profuse bleeding and discharge of cerebro fluid from right ear; right mastoid ecchymosis. Complete deafness of ear and a slight weakness of right side of face of the peripheral type head muscles being involved). Pupils equal but react sluggishly nystagmus. Reflexes—patellar very much increased [illegible] Fundi—retinal veins [illegible] margins of both optic discs [illegible] a slight elevation of nasal half of right optic disc visible. Lumbar puncture—clear cerebrospinal fluid under high pressure (approximately [illegible] mm.).

Treatment.—Expectant palliative. It was thought advisable to observe the patient carefully in the hope that it would be able "to take care of" the condition itself by natural absorption of the [illegible] "wet" condition of the brain as influenced by the increased intracranial pressure. However, within one hour after this examination, patient became very stuporous, drowsy and gradually lapsed into profound unconsciousness 4 hours after admission.

Examination 5 hours after admission.—Temperature, 100°; pulse, 60; respiration, 20; blood-pressure, 160. Unconscious. Bleeding and discharge of cerebrospinal fluid from the right ear has ceased (about 2 hours ago) and yet where the signs of intracranial pressure became more marked—undoubtedly the leakage being a factor in producing the increased intracranial pressure. Pupils contracted equally and do not react to light. Patellar reflexes very active; no ankle clonus but suggestive double Babinski. Abdominal reflexes depressed but equally so. Fundi—retinal veins dilated and tortuous; edematous blurring of both nasal and temporal sides of optic discs but no measurable swelling of the discs themselves.

Treatment.—An immediate right subtemporal decompression advised in order to prevent the more marked signs of medullary compression and possibly medullary edema.

Operation 5 hours after admission.—Right subtemporal decompression; usual vertical incision; bone removed and no complications. Dura very tense and upon incising it, clear cerebrospinal fluid spurted; upon enlarging dural opening, a very "wet" edematous cortex tended to protrude under high pressure. The arachnoid bulged tensely owing to the large amount of clear cerebrospinal fluid beneath it and spurted to a height of 3 inches when tiny small openings were made in the arachnoid—these little "fountains" continuing to spurt to a height of 3 inches for over 2 minutes. Owing to the escape of a large amount of this clear cerebrospinal fluid, the tension of the cortex lessened and it receded so, that at the end of the operation it pulsated almost normally; a bilateral decompression, therefore, was not considered necessary; no hemorrhage nor laceration of the cortex was visible. Usual closure with 2 drains of rubber tissue inserted (the lower drain which was inserted at the base in the middle fossa of the skull between the temporosphenoidal lobe and dura was accidentally pulled out at the end of the operation; an attempt was made to re-insert it down to the cortex but "blindly"). Duration, 55 minutes.

Post-operative Notes.—Twelve hours later: Temperature 101.2°; pulse, 60; respiration, 16; blood-pressure, 160. Twenty-four hours later: Temperature, 101.2°; pulse, 55; respiration, 16; blood-pressure, 160. Thirty hours later: Temperature, 101°; pulse, 50; respiration, 14; blood-pressure, 170. Besides the gradual lowering of the pulse- and respiration-rates, they both became at the last examination rhythmically irregular and of the Cheyne-Stokes character; the decompression area was most tense and very little drainage of cerebrospinal fluid appeared. For fear of an increasing cerebral edema or possible hemorrhage, it was thought advisable to open the decompression wound and this was performed 30 hours after the first operation: the cortex was very tense and "dry," but no hemorrhage was ascertained; the lateral ventricle was tapped, allowing one ounce of blood-tinged cerebrospinal fluid to escape but not under pressure. Rubber tissue drain inserted properly and wound closed in the usual manner. (Condition considered one of temporary edema of a "water-logged" brain.) As the pulse remained between 50 and 56 and at one time descended even to 48, it was then debated as to the advisability of a bilateral decompression; however, as profuse drainage of clear cerebrospinal fluid was now occurring, it was decided to wait for a period of 24 hours; by this time the pulse had ascended to 58 and the general condition of the patient became so much improved that a bilateral operation was not considered as being indicated and the patient made an excellent recovery.

Examination at discharge (13 days after admission and operation).—Temperature, 98.8° ; pulse, 68; respiration, 18; blood-pressure, 142. Patient complains of an occasional frontal headache and "things sometimes get black before my eyes"; general weakness. Patient insisted, however, upon going home where he could be "quiet." Only incomplete right facial paralysis and there is a definite return of hearing of the right ear—bone conduction, however, being greater than air conduction; otoscopic examination discloses a laceration in the inferior portion of right tympanic membrane. Pupils equal and react normally. Reflexes very active but otherwise negative. Fundi—retinal veins enlarged; nasal margins of both optic disks blurred and obscured by edema, but other details of optic disks clear. Decompression area bulges beyond flush of scalp and pulsates. Patient advised to remain very quietly at home; daily catharsis and light diet; no alcohol.

Examination (June 10, 1914—12 months after injury).—Patient complains of an occasional frontal headache; otherwise well and works daily. Decompression area has not "sunken in" possibly as much as it should by this time; normal pulsation, however. No weakness of right side of face can be elicited by special tests; hearing of right ear, however, not as acute as left ear; bone conduction slightly greater than air conduction in right ear. Pupils equal and react normally. Reflexes active, but otherwise negative. Fundi—retinal veins slightly enlarged; all details of optic disks clear and distinct.

Examination (October 20, 1916—40 months after injury).—Patient still has an occasional headache but "it doesn't bother me"; works daily at former occupation. Decompression area depressed and pulsates normally. Reflexes active but otherwise negative. Fundi negative.

Last Examination (July 16, 1918—61 months after injury).—Patient says he still has a headache "once in awhile," but "I have become irritable and cranky"; upon questioning patient, he admits some financial troubles since his marriage last year. Decompression area depressed and slightly smaller owing to new bone formation about its periphery; difficult to palpate any pulsation. Impairment of hearing of right ear still present; bone conduction greater than air conduction. Reflexes active but otherwise negative. Fundi negative.

Remarks.—In going over this case, I feel it would have been better surgical judgment to have performed a bilateral decompression at the time of the injury; the condition was one of severe cerebral edema and the patient was very fortunate not to have been permanently damaged—it was too great a risk not to have performed a bilateral decompression at the time. It will be very interesting to follow this patient for a longer period, as it is difficult to conceive of his not being rendered emotionally unstable at least.

It is possible that the lower drain was not reinserted properly so that little or no drainage could occur; otherwise, if excellent drainage had occurred it is very probable that this secondary increase of intracranial pressure following the operation would not have happened. This lower drain inserted into the middle fossa between the inferior surface of the temporosphenoidal lobe and the underlying dura affords excellent drainage of hemorrhage or cerebrospinal fluid collected in the middle fossa—a large cistern at the base of the skull; it is very important in these patients that this drain be placed accurately in order to insure efficient drainage.

It was a very significant observation that the signs of an increasing intracranial pressure occurred rapidly following the cessation of the discharge of blood and cerebrospinal fluid from the right ear; to a large extent, this patient was "decompressing" himself by means of this channel of escape, but it is doubtful whether this amount of drainage would have been of sufficient quantity to have lessened the high increased intracranial pressure of this patient to such an extent that the operation of subtemporal decompression and drainage could have been avoided. This escape of blood and cerebrospinal fluid through a line of fracture into the ear rarely persists longer than 48–60 hours, and if it should, then the danger of infection of the ear and a possible meningitis becomes greater daily; repeated lumbar punctures may be advisable in these patients if the increased intracranial pressure is not extreme and in this way the discharge from the ear can be stopped.

The impairment of hearing is undoubtedly a permanent one due to the formation of scar tissue in the tympanic membrane and about the ossicles together with their retraction, and thus the bone conduction will always be greater than the air conduction; fortunately, in these traumatic cases the impairment of hearing is usually only a temporary one, unless the internal ear and the auditory nerve itself has been damaged.

The temporary facial paralysis of the peripheral type was undoubtedly due to a mild edematous compression of the facial nerve at the usual site—its narrow bony canal, the aqueduct of Fallopius. Unless the facial nerve itself is severed or firmly compressed by bone, then the facial paralysis of these patients is of only temporary duration.

CASE 53.—Acute severe brain injury; signs of high intracranial pressure due to cerebral edema. Right subtemporal decompression and drainage. Excellent recovery.

No. 651.—Fabian. Twenty-three years. White. Single. Laborer. Poland

Operation January 12, 1916—6 days after injury. Right subtemporal decompression and drainage.

Discharged March 1, 1916—48 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While riding a horse to work, patient was thrown, striking his head against a rock; immediate loss of consciousness; brought to the hospital in ambulance.

Examination upon admission (50 minutes after injury).—Temperature, 97.8°; pulse, 132; respiration, 38; blood-pressure, 104. Unconscious and in extreme shock. Laceration of scalp over right eye. No bleeding from nose, mouth or ears. Superficial examination reveals the reflexes absent while the pupils are dilated and non-reacting to light.

Treatment.—Vigorous anti-shock measures instituted: external warmth being most important—the blankets of the bed having been thoroughly warmed and at least 6 hot-water bottles applied to body (to both feet, between the thighs, in the axillæ, etc.); hot black coffee enemata; quiet. Patient gradually emerged from the condition of shock after 4 days and 2 days later, after being in a semiconscious condition, patient began to show the signs of an increasing intracranial pressure.

Examination (6 days after admission).—Temperature, 101.2°; pulse, 64; respiration, 16; blood-pressure, 140. Patient has become increasingly difficult to arouse, although his general condition is fair; takes liquid by mouth; bowels move daily. Otoscopic examination negative. Pupils equal and react normally. Reflexes—patellar very much exaggerated but equal; double exhaustible ankle clonus and suggestive Babinski; abdominal reflexes depressed. Fundi—retinal veins have become dilated and the nasal halves of both optic disks have become entirely obscured, as well as the temporal margins which before had been clear and distinct. Lumbar puncture—slightly blood-tinged cerebrospinal fluid under high pressure (approximately 18 mm.). X-ray (Doctor A. J. Quimby)—"multiple lines of fracture of right squamous area of vault" (Fig. 77).

Treatment.—An immediate right subtemporal decompression and drainage was now advised in the belief that to delay longer would risk not only the life of the patient but also his future good health.

Operation (6 days after admission).—Right subtemporal decompression: usual vertical incision, bone removed and no complications; irregular line of fracture of squamous portion of right temporal bone bifurcating anteriorly into right frontal and superiorly into right parietal bones. Dura moderately tense and upon incising it, clear cerebrospinal fluid welled out under moderate pressure; upon enlarging dural opening, a moderately "wet" edematous cortex tended to protrude but did not rupture and it very quickly receded owing to the loss of cerebrospinal fluid. No subdural hemorrhage nor cortical laceration ascertained. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Patient made an excellent operative recovery, but he remained in a mild state of mental confusion and disorientation for a period of several weeks, so much so that it was finally decided to transfer the patient to Bellevue Hospital, where he could remain for a period of months until he might regain his normal condition; incision healed *per primam.*

Examination at discharge (54 days after admission and 48 days after operation).—Temperature, 99°; pulse, 78; respiration, 22; blood-pressure, 136. No complaints—"I feel all right." Patient, however, has a definite mental confusion and also retardation with a moderate degree of cortical irritability, as disclosed in his restlessness, inability to sleep and emotional instability. Decompression area slightly depressed; pulsates normally. Pupils equal and react normally. Reflexes very active but otherwise negative. Fundi—retinal veins slightly enlarged but no edema of optic disks.

Last Report (Augunst 20, 1918—19 months after injury).—Patient is in the army and has been in good health during the past year. It was, however, 9 months after the injury before the patient "became himself again." (A further report regarding this patient will be made if he can be located later.)

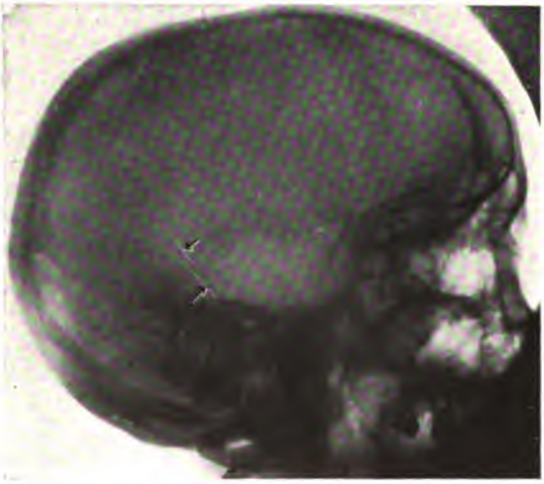


FIG. 77.—Two oblique and parallel linear fractures of the posterior portion of the right squamous bone in a patient having a high intracranial pressure due to cerebral edema alone; right subtemporal decompression and drainage permitted an excellent recovery.

Remarks.—The advisability of waiting until the signs of severe shock had disappeared is very evident in this case; if an operation had been performed during the period of severe shock—no matter how badly the brain had been injured, the risk of such an operation at that time would have been very great indeed; besides, during the period of severe shock there were no signs of a marked increase of the intracranial pressure and therefore no indication for the operation of decompression—a marked increase of the intracranial pressure being the only indication for advising a cranial decompression upon these patients; naturally all cases of depressed fractures of the vault are not included in this grouping as they should all be operated upon—the depressed area being elevated or removed. I feel, however, in this particular case, that the operation of decompression should have been advised earlier—at least 24 hours—and possibly 48 hours before, and it is possible that the post-operative mental and emotional disturbances could have been lessened, if not entirely avoided.

It is rather surprising that there was no impairment of the right middle ear in this patient due to a possible line of fracture extending into the right petrous bone. The röntgenogram indicated several lines of fracture extending through the right squamous bone and beyond which was confirmed at the operation; usually in these patients having fractures of the squamous portion of the temporal bone, the line of fracture extends downward into the middle fossa and into the petrous bone adjacent to the middle ear and thus frequently causing a rupture of the tympanic membrane and the discharge of blood and cerebrospinal fluid into the middle ear. In this patient, however, neither was there blood blocked in the middle ear, as the otoscopic examination was negative, nor was the tympanic membrane lacerated. It would seem, therefore, that the tympanic cavity had escaped the line of fracture in this case, if the fracture did descend into the middle fossa of the skull; the fact that there was no right mastoid ecchymosis would confirm this opinion in addition to the fact that the hearing was not impaired.

It is very instructive to follow a patient of this character who has regained completely his normality as before the injury; no doubt, a certain percentage of these patients would have recovered their lives and also have regained even their normal condition as before the injury without any operation having been performed upon them; but it is indeed much safer to perform the operation of subtemporal decompression under the proper conditions of assistance and asepsis upon these patients early and thus avoid not only the danger of medullary compression and possibly edema, and thus the death of the patient, but also in a larger number of them to avoid those post-traumatic conditions so common in patients following brain injuries, where the increased intracranial pressure has not been relieved by an earlier cranial operation. Besides, it is impossible to ascertain just which patients will recover not only their former normality but even their lives at the time of the injury, and therefore in cases of doubt, I feel it is wiser to advise the operation as a certain means of lessening the intracranial pressure rather than to depend upon the means of natural absorption to lessen the increased intracranial pressure, and then if it should not, it is frequently too late not only to obtain a normal individual but also to cause even a recovery of life itself. A study of the statistics regarding these data in this series of patients confirms this opinion.

It will be observed that a number of patients in this series of acute brain injuries with early operation that X-ray pictures were taken in only a small number of them; this is due to the fact that most of these patients were in a very critical condition after their admission to the hospital and as the X-ray picture is of no importance in their treatment (except when it is a question of a depressed fracture of the vault), röntgenograms were taken in only a few of the patients; also, as most of these patients were discharging blood and cerebrospinal fluid from either or both ears, a fracture of the skull was known to be present, but as is well known the important factor to be ascertained is not the presence or not of a fracture of the skull, whether of the vault or of the base, but the presence or not of a marked increase of the intracranial pressure, which can be accurately ascertained by the careful tests as described in these patients. It is to be regretted, however, that röntgenograms were not made of all of the skulls of these patients during their convalescence at least and as a matter of record alone; this should be done in all cases of brain injuries and possible fractures of the skull.

Case 54.—Acute severe brain injury; signs of high intracranial pressure due to cerebral edema. Left subtemporal decompression and drainage. Excellent recovery.

No. 679.—Thomas. Twenty-six years. White. Single. Clerk. Poland.

Admitted March 25, 1916, Nassau Hospital, Mineola. Referred by Doctor L. A. Van Kleeck.

Operation March 28, 1916—72 hours after injury. Left subtemporal decompression and drainage.

Discharged April 20, 1916—22 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While coasting down a hill, patient was struck by an automobile; immediate loss of consciousness; brought to the Nassau Hospital in the automobile, and upon arrival patient was in a moderate degree of shock—temperature, 98°; pulse, 108; respiration, 28; blood-pressure, 116. Semiconscious but could be aroused by firm supraorbital pressure. No bleeding from nose, mouth or ears. Pupils enlarged but equal and react sluggishly to light. Reflexes active but otherwise negative. Patient gradually recovered from the condition of shock; the pulse descended to 70, the blood-pressure increased to 130 and patient became more conscious, but could not speak at any time. As the signs of intracranial pressure were increasing, a consultation was now considered advisable.

Examination (March 28, 1916—70 hours after injury).—Consultation with Doctor Van Kleeck: Temperature, 99.8°; pulse, 60; respiration, 18; blood-pressure, 142. Conscious but drowsy—could be aroused but would quickly lapse into unconsciousness. Unable to speak a word when conscious—a condition of pure motor aphasia; no agraphia; no astereognosis. Definite weakness of right side of body, particularly of lower right side of face (cortical type of facial paralysis in that the forehead muscles were not involved). Pupils equal and react normally. Reflexes—patellar exaggerated, right being possibly greater than left; suggestive right Babinski; abdominal reflexes—right depressed. Fundi—retinal veins dilated and tortuous; both nasal halves and temporal halves of optic disks obscured but no measurable swelling—that is, not a "choked disk," but a papilledema of early degree. Lumbar puncture—clear cerebrospinal fluid under high pressure (approximately 18 mm.).

Treatment.—An immediate left subtemporal decompression advised to lessen the increasing intracranial pressure; there was no bleeding or discharge of cerebrospinal fluid through the nose or ears, which is usually very helpful in lessening a moderate increase of the intracranial pressure, while the risk of infection and a resulting meningitis are comparatively of rare occurrence with proper prophylactic measures. Before preparing the patient for operation, the accompanying photographs were taken (Figs. 78–81).

Operation (72 hours after admission).—Left subtemporal decompression: usual vertical incision, bone removed and no complications. Dura very tense and upon incising it, a slightly straw-colored cerebrospinal fluid spurted a distance of 3 inches; upon enla[illegible]dural opening, a very congested cortex was exposed, being very "wet" and edematous, particularly beneath the Sylvian fissure; large quantities of cerebrospinal fluid escaped and thus permitted the brain to recede so that the cortical protrusion did not rupture and normal pulsation occurred before the end of the operation. No punctate cortical hemorrhage nor lacerations were visible—merely a slightly blood-tinged subarachnoid cerebrospinal fluid which continued to "sweat" throughout the operation. Usual closure with 2 drains of rubber tissue inserted. Duration, 50 minutes.

FIG. 78. FIG. 79.

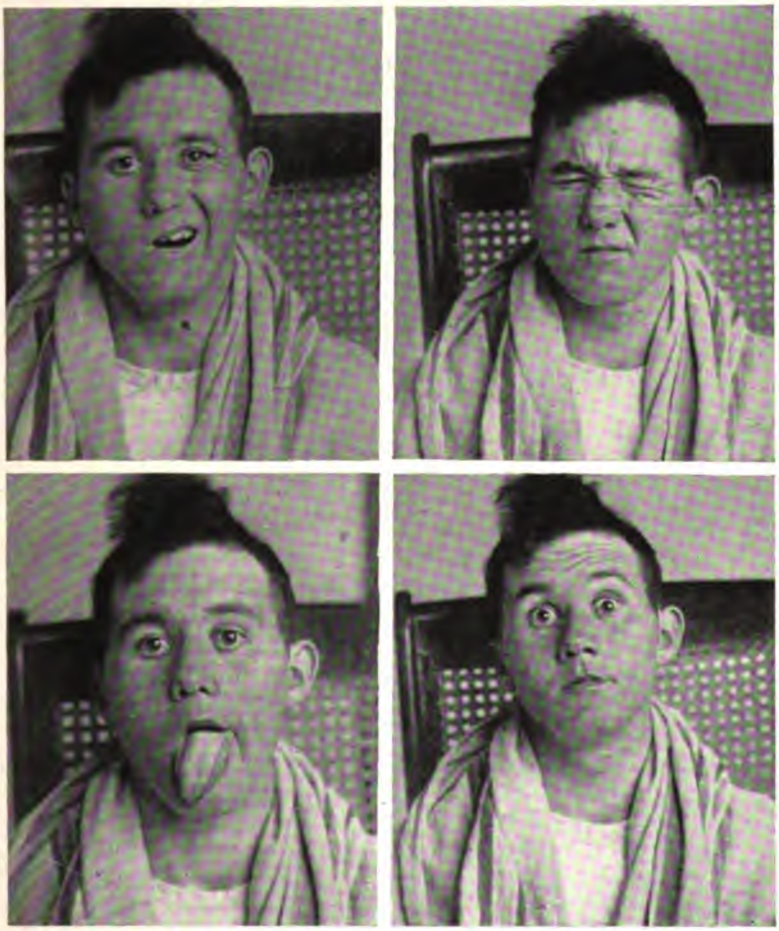


FIG. 80. FIG. 81.

FIGS. 78–81.—Right facial paralysis of the cortical central type due to an extensive cerebral edema of the left cortical hemisphere; an excellent recovery obtained by means of a left subtemporal decompression and drainage. Note the ability of the patient to corrugate the muscles of the right half of the forehead, and to close the right eye—thereby differentiating this central type of paralysis from the peripheral type, due to a lesion of the facial nerve itself. The deviation of the tongue to the right is well illustrated.

Post-operative Notes.—Uneventful operative recovery: within 24 hours after operation, patient became more conscious than at any time since the injury, the pulse ascended to 76 and the weakness of the right side of the face practically disappeared. Patient began to use monosyllables on the fourth day after operation and then his speeeh rapidly improved.

Examination at discharge (25 days after admission and 22 days after operation).—Temperature, 98.6°; pulse, 78; respiration, 20. Perfectly conscious. No complaints except for a dull headache. Some slight impairment of speech elicited by the test phrases (patient being Polish and not speaking English well, an accurate estimate of his speech impairment was very difficult). No weakness of right side of body ascertained. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi negative, except for a slight enlargement of the retinal veins. Decompression area does not bulge beyond the flush of scalp; normal pulsation.

Last Report (August 17, 1918—29 months after injury).—No complaints; patient works daily. No impairment of speech and no weakness of the right side of body apparent to patient. In excellent condition both mentally and physically.

Remarks.—It is interesting to note that there was no large hemorrhage present over the cortex of the motor speech area or the face area involved—there being only a very "wet" edematous cortex with congested supracortical vessels. This would tend to confirm the belief that a localized cortical edema was the cause of the speech impairment and of the weakness of the right side of the face, so that, as soon as a decompression was performed and proper drainage instituted for this "pent up" cerebrospinal fluid, the condition quickly disappeared and an excellent recovery was obtained—there being no definite organic lesion which would produce a permanent impairment. If there had been a fracture through the left petrous bone so that the cerebrospinal fluid could have escaped through this line of fracture and through a torn tympanic membrane out into the external auditory canal, then this patient would have had an excellent chance of "decompressing" himself and thus the risk of an operation would have been avoided.

The condition of pure motor aphasia is a most infrequent one and yet this patient at the examination before operation appeared to have it, in that when conscious, he was able to understand both spoken and written words, and could write words and answers in his own language (Polish), but he was unable to speak either spontaneously or by repeating words either in English or Polish; there was no apraxia. That is, in the case of a box of matches he was able to write "matches," upon being asked what they were; he was able to pick up a match from a number of objects when requested to do so, and upon being asked what they were used for, he went through the motion of striking a match, and then wrote, "to light the fire"; with his eyes closed, patient was able to write down afterward the names of various objects which had been placed in his hand, therefore, there was no astereognosis present. From the other examinations also, it would seem that this patient had an uncomplicated condition of motor aphasia, so that at the operation of left subtemporal decompression (his parents and grandparents all being right-handed), a definite lesion and most probably a hemorrhage was anticipated in the cortex of the posterior portion of the third left frontal convolution—Broca's motor speech area; only an extreme edematous con-

dition of this area of the cortex was observed—an acute cerebral edema—and this observation was confirmed by the rapid subsidence and disappearance of the motor aphasia following the operation of decompression and drainage.

The right facial paralysis of the cortical type in that the right forehead muscles were not involved was undoubtedly due to the same cause—an acute cortical edema following the injury; the almost immediate recovery from this facial weakness after the operation of decompression and drainage would also confirm the diagnosis of acute cortical edema—at least of the left cerebral cortex. The immediate disappearance of the left hemiplegia following operation, when only a "wet" edematous cortex was revealed at operation, would tend to confirm the opinion that localized cerebral edema—or rather cerebral edema more acute in one portion of the brain than in another—is a definite factor and possibly the only factor in some patients in producing a definite paralysis; repeated lumbar punctures to improve the condition of this patient would have been very interesting and instructive.

Case 55.—Acute severe brain injury; signs of high intracranial pressure due to cerebral edema. Left subtemporal decompression and drainage. Excellent recovery.

No. 564.—Florence. Fourteen years. White. School. U. S.

Admitted May 2, 1916, Polyclinic Hospital.

Operation May 3, 1916—90 hours after injury. Left subtemporal decompression and drainage.

Discharged May 17, 1916—14 days after operation.

Family history negative.

Personal history negative.

Present Illness.—Three days ago while patient was playing in the street, a brick fell a distance of 4 stories, striking her upon the head; immediate loss of consciousness and she did not become semiconscious until 2 hours later; very dizzy but still able to walk home; since then, patient has had severe continuous headache and dizziness; brought to the hospital in an automobile.

Examination upon admission (70 hours after injury).—Temperature, 99.6°; pulse, 70; respiration, 20; blood-pressure, 124. Well-developed and nourished. Conscious, but rather drowsy and complains of severe frontal and occipital headache; keeps her eyes closed and feels less dizzy by doing so. No bleeding from nose, mouth or ears. A large hematoma—the size of an egg, over the posterior portion of the left parietal bone. Pupils equal and react normally. Reflexes very active but otherwise negative. Fundi: retinal veins engorged and tortuous in places obscured by edema; papilledema of 2 diopters—both optic disks being entirely obscured by the edema—that is, almost to the degree of "choked disks." Lumbar puncture—clear cerebrospinal fluid under high pressure (approximately 20 mm.). X-ray (Doctor W. H. Stewart)—"shows a sagittal fracture extending posteriorly downward into the occipital bone; then forward on the right side into the posterior fossa, running anteriorly into the base back of the mastoid cells."

Treatment.—An immediate left subtemporal decompression advised to lessen the intracranial pressure in the belief that a medullary compression might be avoided; the ophthalmoscopic examination revealing practically a "choked disk" in each fundus was very impressive and removed this patient immediately from that large group of patients in whom the expectant palliative treatment is sufficient. Consent for the operation was not obtained until following day.

Operation (90 hours after injury).—Left subtemporal decompression; exploratory scalp incision of hematoma. Usual incision, bone removal and no complications. Dura exceedingly tense; upon incising it, slightly straw-colored cerebrospinal fluid spurted to a height of 1 foot, striking the operator in the eye; upon enlarging the dural opening, a very "wet" edematous cortex tended to protrude, but owing to the rapid loss of much cerebrospinal fluid (slightly straw-colored), the cortex did not rupture and its tension soon lessened so that it was pulsating at the end of the operation. No cortical hemorrhage or laceration ascertained. Usual closure with 2 rubber tissue drains inserted. Small vertical scalp incision now made over the hematoma; a small subcutaneous blood-clot evacuated, but upon retraction no fracture of the underlying bone revealed; incision loosely sutured with one drain of rubber tissue inserted. Duration, 60 minutes.

Post-operative Notes.—Uneventful operative recovery; patient was conscious and rational within 24 hours and did not complain of headache except for soreness at the site of operation; pulse ascended to 86, and at the end of 48 hours the nasal halves only of both optic disks were blurred. Both incisions healed *per primam.*

Examination at discharge (18 days after injury and 14 days after operation).—Temperature, 98.8°; pulse, 82; respiration, 24; blood-pressure, 126. No complaints. Decompression area bulges slightly beyond the flush of scalp; normal pulsation. Hearing negative; otoscopic examination negative. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—retinal veins enlarged; upper nasal sector of margins of both optic disks blurred and indistinct from edema.

Examination (September 20, 1917—16 months after injury).—No complaints; patient was able to return to school 4 months after the injury and has been perfectly well. Decompression area slightly depressed and pulsates normally. Reflexes active but otherwise negative. Fundi negative.

Last Examination (August 22, 1918—27 months after injury).—No complaints; patient has been working this summer as a salesgirl. Decompression area depressed and pulsates normally. Reflexes negative. Fundi negative.

Remarks.—It is rare for a patient with an acute brain injury to have a papilledema to the extent of "choked disks" and yet with so few signs of an increased intracranial pressure as this patient; the headache was severe but there were practically no signs of a medullary compression which might be expected in an acute intracranial condition having so high an increased pressure as to produce a papilledema of 2 diopters and to register approximately 20 mm. of pressure at lumbar puncture. It seems to be, however, merely another illustration of the facility with which high intracranial pressure in children is withstood, and conversely the great difficulty for increased intracranial pressure to produce the signs of medullary compression, however mild, in children around the age of 12 years and younger; not only can these patients withstand safely a higher intracranial pressure than can adults but their powers of natural absorptioon, are much greater, and therefore the treatment of these patients from an operative standpoint can be much more conservative than that of adults where the danger of high intracranial pressure in acute conditions is most grave.

The presence of a small amount of blood in the cerebrospinal fluid in this patient was of such little quantity that it could not in itself have produced, I believe, any untoward symptoms or signs, so that the intracranial pressure was due almost entirely to an excess of the cerebrospinal fluid producing this so-called "cerebral edema."

The hematoma over the posterior portion of the left parietal bone was incised in order to ascertain the presence or not of an underlying fracture of the bone; if depressed, then it could be elevated or removed, and if merely a linear fracture was present then the hematoma could be evacuated and drained, and thus the danger of an infection of the hematoma extending downward through the line of fracture and producing a meningitis and its complications of brain abscess, etc., could be thus avoided. No fracture of the underlying bone being ascertained, however, the hematoma itself was merely evacuated, drained and healing *per primam* resulted. The skin overlying the hematoma was rather bruised and contused, and these are just the cases where an underlying hematoma becomes infected very easily, and if there is present an underlying fracture of the skull and the infected hematoma is not drained early, most serious consequences intracranially may occur. If it is definitely known that there is no underlying fracture of the vault, then in certain selected patients the hematoma may be aspirated through a "clean" area and thus the danger of an infection of the hematoma is also lessened. I have had several patients die from an infection of a simple hematoma of the scalp with and, in one case, without a fracture of the underlying vault, and this disastrous result is most impressive.

The röntgenograms of this patient show a fracture of the skull in a most dangerous part of the skull—extending subtentorially and near the foramen magnum and across the lateral sinus. The danger of an acute medullary compression with its resulting medullary edema is very great in this type of cranial injury, and especially in adults; children are not so susceptible to an acute medullary compression and therefore the prognosis, with and without operation, upon children is usually much better than in the older patients.

Case 56.—Acute severe brain injury; marked signs of high intracranial pressure due to cerebral edema. Right subtemporal decompression and drainage. Recovery.

No. 587.—David. Twenty-six years. White. Single. Taxi-driver. U. S.
Admitted May 24, 1916, Polyclinic Hospital.

Operation June 12, 1916—18 days after injury. Right subtemporal decompression and drainage.

Discharged June 24, 1916—12 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While driving his taxicab, patient was struck by an automobile; immediate loss of consciousness; brought to the hospital in the automobile.

Examination upon admission (35 minutes after injury).—Temperature, 98.4°; pulse, 92; respiration, 18; blood-pressure, 138. Unconscious, but can be aroused by firm supraorbital pressure. Large hematoma over the right occipital area. No bleeding from nose, mouth or ears; no orbital nor mastoid ecchymoses. Pupils slightly enlarged but react normally. Reflexes present and equal; no Babinski. Fundi negative. Lumbar puncture—clear cerebrospinal fluid under very slightly increased intracranial pressure (approximately 10 mm.).

Treatment.—Expectant palliative; only a mild degree of shock is present and at this time, the patient was considered as having the condition of cerebral concussion and the prognosis was good. Within 20 hours patient became sufficiently conscious to be able to talk rationally; he remained, however, in a very apathetic, drowsy condition—sleeping most of the time and without marked signs of high intracranial pressure for a period of 2 weeks; no abnormality of the reflexes noted. Fundi showed signs of mild intracranial pressure, although at 2 different lumbar punctures the cerebrospinal fluid was not under high intracranial pressure; the pulse, however, had descended to 60 and had remained thereabouts during the 2 weeks. Patient did not show a marked improvement during this period and remained in a drowsy lethargic condition—neither improving nor becoming markedly worse until June 11, 1916 (17 days after admission), when the patient became definitely worse and the signs of a marked increase of intracranial pressure appeared.

Examination (17 days after admission).—Temperature, 99.2°; pulse, 58; respiration, 16; blood-pressure, 140. Very stuporous but can be aroused by firm supraorbital pressure. Pupils equal but react rather sluggishly. Reflexes—patellar present and equal; no Babinski nor ankle clonus; abdominal reflexes equal but rather depressed. Fundi (Doctor J. A. Kearney)—"retinal veins very much enlarged; both optic disks completely obscured by edema—the elevation of tissue being 2 diopters plus; fundi about disks hemorrhagic and vessels buried in the new tissue formation." Lumbar puncture—clear cerebrospinal fluid under high intracranial pressure (approximately 22 mm.); Wassermann test negative.

Treatment.—An immediate right subtemporal decompression now advised to lessen this sudden increase of the intracranial pressure; consent, however, for the operation was not obtained until 20 hours later.

Operation (18 days after injury and admission).—Right subtemporal decompression: usual vertical incision, bone removed and no complications. Dura thickened, tense and very vascular; upon incising it, clear cerebrospinal fluid welled out, and upon enlarging the dural opening a very "wet" edematous cortex protruded, but did not rupture owing to the rapid escape of a large quantity of cerebrospinal fluid. Cortex showed throughout many old punctate hemorrhages—being very similar to the cross section of spleen; about the vessels in the sulci there was a subarachnoid thickening and whitish induration as though due to the organization of a former subarachnoid hemorrhage. The cortical tension was so high at the very beginning of the operation that the lower margin of the third temporal convolution ruptured for a distance of one-half inch. Brain pulsated normally at end of operation. Usual closure with 2 drains of rubber tissue inserted. Duration, 55 minutes.

Post-operative Notes.—Within 24 hours after operation, patient became more conscious and rational than at any time following the injury; the pulse, however, remained between 60 and 66 for a period of 5 days and then ascended to 70; decompression area bulged very tensely for 5 days, and as it lessened its protrusion, the fundi cleared so that the temporal margins of both optic disks became visible.

Examination at discharge (30 days after admission and 12 days after operation).—Temperature, 99°; pulse, 70; respiration, 18; blood-pressure, 134. Rational and yet a definite mental retardation—both in thinking and talking. No complaints, except "I want to sleep all the time." Decompression wound bulges slightly beyond the flush of scalp; pulsation normal. Pupils equal and react normally. Reflexes present and equal and no abnormality. Fundi (Doctor J. A. Kearney)—"nasal margins of both optic disks not clear; surrounding retinæ rather congested and suffused; retinal vessels possibly a little enlarged."

Treatment.—Patient insisted upon going home; his relatives were advised to keep him at home and as quietly as possible.

Examination (April 20, 1917—11 months after injury).—Patient has made an unexpected and unusually good recovery. No complaints and works daily, driving a hansom-cab; is becoming, however, more and more alcoholic with marked tremor of both hands. Apparent retardation of speech, although his mother states the patient was always a little "slow" and not so "quick" as her other sons. Pupils equal and react normally. Reflexes present and equal. Fundi rather congested and suffused throughout entire retinæ, but no obscuration of details of optic disks. Decompression area slightly depressed and pulsates normally.

Last Examination (August 4, 1918—26 months after injury).—No complaints referable to head injury; patient, however, is in his usual condition of mild intoxication, but always able to perform his work as a driver without accident. Decompression area only slightly depressed and pulsates normally. Reflexes negative. Fundi still rather congested and suffused, but no blurring of the details of optic disks. Urine examination—small trace of albumen with an occasional hyaline and granular cast.

Remarks.—The condition of this patient was a most puzzling one in that, at operation almost 3 weeks after the injury, the signs of definite subarachnoid and even cortical punctate hemorrhages were found, and yet it was not until 17 days after the injury that the signs of a high increase of intracranial pressure appeared; undoubtedly, it was a case of delayed cerebral edema superimposed upon the condition of subarachnoid, and cortical punctate hemorrhages—the latter in themselves not being of sufficiently large amount to produce signs of a marked increase of the intracranial pressure. Undoubtedly there are many similar latent cerebral conditions with hemorrhage following head injuries which are not recognized but are considered to be merely conditions of cerebral edema, and then when the patient does not make such a good recovery as would be expected from a condition of simple cerebral concussion, then the condition is considered one of post-traumatic neurosis and so labelled. If this patient had been in better physical condition at the time of the injury (particularly in regard to alcoholism and his general resistance), he might have been able himself to have "taken care of" the intracranial condition by means of simple absorption, and thus have prevented the onset of an acute cerebral edema which was the immediate cause of the high intracranial pressure and thus rendering a decompression imperative to avoid a marked medullary compression and possibly medullary edema. In patients having similar conditions, naturally no operation can be advised unless there are definite signs of an increased intracranial pressure; it might be argued that a decompression with drainage would aid in the natural absorption of the subarachnoid hemorrhage and possibly the cortical punctate hemorrhages, and yet this condition cannot be with certainty diagnosed, and if there is no increased pressure naturally a decompression cannot be advised; I believe that the great majority of these patients having similar conditions make excellent recoveries without any operation; if, however, the signs of an increased intracranial pressure do occur, then the operation of decompression and drainage should be immediately advised. This condition of latent cerebral edema following brain injuries occurs most frequently in alcoholics, and it is the cause of the high mortality of brain injuries in this type of patient—the acute cerebral edema occurring earlier in most of the patients than in this case. This patient is also interesting in that the cerebrospinal fluid removed by lumbar puncture was clear each time, and yet there was present intracranially subarachnoid bleeding; this observation tends to confirm the experimental work of Doctor Norman Sharpe regarding the direction of the flow of the cerebrospinal fluid which flows up from the fourth ventricle over the cortex first rather than from the ventricle down into the spinal canal; if, however, the supracortical hemorrhage is of large amount then the blood appears in the spinal cerebrospinal fluid without difficulty.

CASE 57.—Acute severe brain injury; marked signs of extreme intracranial pressure due to a high and increasing cerebral edema. Bilateral decompression and drainage. Excellent recovery.

No. 166.—Walter. Thirty-two years. White. Married. Reporter. U. S. Admitted May 6, 1914, Polyclinic Hospital. Referred by Doctor J. A. Bodine.

Operations May 9, 1914—3 days after injury. Bilateral decompression and drainage.

Discharged May 19, 1914—10 days after operations.

Family history negative.

Personal history negative.

Present Illness.—While crossing the street, patient was struck by an automobile; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (50 minutes after injury).—Temperature, 97.4°; pulse, 144; respiration, 6 (?); blood-pressure, 104. Well-developed and nourished. Unconscious and in condition of extreme shock—cold, clammy skin, irregular weak pulse and respiration scarcely perceptible. No bleeding from nose, mouth or ears. No examination other than this was made on account of the severity of the shock, although it was noted that the pupils were widely dilated and did not react; patient was considered moribund.

Treatment.—Vigorous anti-shock measures instituted: patient immediately placed in a warm bed (blankets having been themselves warmed), hot-water bottles distributed as follows—2 bottles for feet, one for each side of chest under arm-pit, one upon abdomen with palms of both hands resting upon it and one between the thighs; 6 ounces of hot black coffee per rectum and repeated every 2 hours for 4 times and then hot rectal saline every 4 hours substituted; absolute quiet. After 6 hours, patient improved in his general condition and the shock became less marked—pulse descended and became more regular and full, respiration became more perceptible and regular; patient became semiconscious and restless.

Examination (72 hours after admission).—Temperature, 100°; pulse, 56; respiration, 18; blood-pressure, 146. Patient conscious but confused mentally; the pulse has gradually descended to 56. No bleeding from the ears; no mastoid ecchymoses. Pupils—left larger than right and reacts sluggishly to light. Reflexes: patellar—right greater than left; no ankle clonus but right Babinski; abdominal reflexes—right depressed but still can be elicited. Fundi—retinal veins dilated, tortuous and buried in the edematous retinæ, especially about the disks; both optic disks entirely obscured by edema and there is a papilledema of 2 diopters—that is, an edematous swelling of the optic disks equalling the first stage of "choked disks." Lumbar puncture—bloody cerebrospinal fluid under high intracranial pressure (22 mm.). X-ray (Doctor A. J. Quimby)—"definite oblique line of fracture extending downward from right parietal bone into right squamous bone" (Fig. 82).

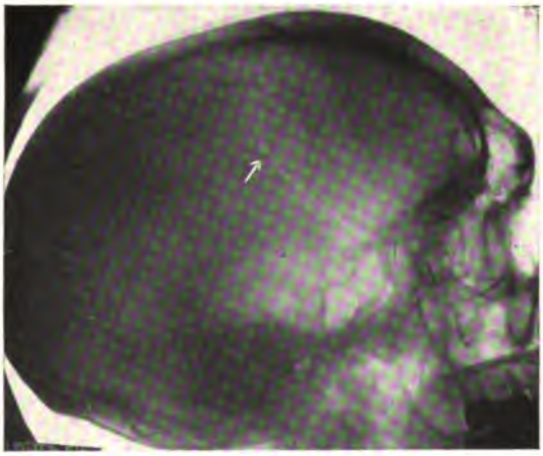


FIG. 82.—Indefinite linear fracture of the right vault extending into the squamous portion of the right temporal bone. The extreme intracranial pressure due to cerebral edema alone, successfully relieved by a bilateral subtemporal decompression.

Treatment.—An immediate left subtemporal decompression advised in the hope that it might prevent the onset of severe medullary compression.

First Operation (May 9, 1914—74 hours after admission).—Left subtemporal decompression: usual vertical incision, bone removed and no complications. Dura exceedingly tense and bulging; upon incising it, clear cerebrospinal fluid spurted to a height of 1 foot and upon enlarging the dural opening, the bulging temporo-sphenoidal lobe tended to protrude and portions of it did "ooze out"—especially from its lower half; upper portion of temporal lobe was also lacerated by this herniation; no free subdural blood observed but much clear cerebrospinal fluid escaped; the cortex appeared "waterlogged," bulged into opening and did not pulsate. Owing to this high cerebral pressure which could not be relieved by the one operation, a right subtemporal decompression was considered necessary. Usual closure with 2 drains of rubber tissue inserted. Temporary sterile gauze dressing applied.

Second Operation.—Right subtemporal decompression: usual vertical incision, bone removed and no complications; much free blood among the fibres of the temporal muscle beneath the temporal fascia and therefore a fracture of the underlying bone was to be expected, and it was found—being an oblique line of fracture extending from the right parietal bone downward through the upper portion of the underlying squamous bone. Upon removing the bone, a small extradural layer of blood-clot—one-eighth inch in thickness—extended beyond the borders of the decompression opening and it was evacuated. Dura very tense and bulging, and upon incising it, clear cerebrospinal fluid again spurted but only to a height of 4 inches; upon enlarging the dural opening, the underlying cortex bulged but did not rupture; no lacerations evident. Much clear cerebrospinal fluid escaped, allowing the cortex to recede slightly and to pulsate almost normally. Usual closure with 2 drains of rubber tissue. Duration, 85 minutes.

Post-operative Notes.—Patient became conscious 12 hours after operation, pulse ascended to 76, and although during the following 6 days patient was slightly confused mentally, irritable and restless, yet he made an uneventful recovery so that he insisted upon "going home" 10 days after the operation; incisions healed *per primam.*

Examination at discharge (13 days after admission and 10 days after operation).—Temperature, 98.8°; pulse, 80; respiration, 20; blood-pressure, 128. Patient says he feels well, although "I have a little pain in my forehead at times." Some mental retardation evident though no confusion, and patient is well oriented as to time, place and personality. Loss of memory for events of day preceding the injury (retroamnesia)—as is very common. No aphasia nor paraphasia can be elicited by the various tests. Both decompression areas bulging beyond the flush of scalp, but normal pulsation visible and palpable. Pupils equal and react normally. Reflexes: patellar very active—right possibly greater than left; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi—retinal veins enlarged; nasal halves of both optic disks obscured by edema, but temporal halves of disks clear and distinct.

Treatment.—Patient insisted upon returning home; besides the general hygienic measures, he was strongly advised to remain at home quietly and to attempt no work for a period of 3 months, at least.

Examination (July 7, 1914—2 months after injury).—Temperature, 98.8°; pulse, 80; respiration, 20; blood-pressure, 134. No complaints other than a "sort of fulness in the head"; rather irritable at times and wants to return to work. Patient can now remember some of the events of the day preceding the accident (the usual recovery of memory that occurs in these patients). Both decompression areas slightly depressed; normal pulsation. Reflexes active but otherwise negative. Fundi—slight haziness along the lower nasal sector of both optic disks but the retinal veins are of normal size.

Examination (September 20, 1916—28 months after injury).—Except that patient becomes more easily fatigued than formerly and then has an occasional headache, he has no complaints; works daily as a reporter and is apparently very successful. Both decompressions "sunken in"; normal pulsation. Reflexes active but otherwise negative. Fundi negative.

Last Examination (October 4, 1918—55 months after injury).—No complaints. "As strong as ever." Both decompression areas depressed and their diameters have lessened owing to new bone formation about the periphery; only slight pulsation can be palpated. Reflexes active but otherwise negative. Fundi ngeative.

Remarks.—This patient has been a most instructive case: the value of waiting until the signs of severe shock have disappeared before even a thorough examination is made and, by all means, the inadvisability of any operation during this period, are clearly illustrated; this patient would undoubtedly have died if an operation had been attempted during the period of extreme shock. The value of a bilateral decompression is also illustrated, as it is doubtful if the intracranial pressure of this patient could have been sufficiently relieved by a unilateral decompression alone; however, if it had been realized (and it can be more accurately ascertained now by using the spinal mercurial manometer) that the intracranial pressure was so high, then a right subtemporal decompression (the patient being right-handed) would have been advised first to be followed by a left subtemporal decompression—even though the signs indicated (and they did in this patient) a more marked lesion of the left hemisphere; by performing a right subtemporal decompression first in these patients, there is less danger of possible operative trauma producing its clinical signs—the right temporo-sphenoidal lobe being less highly developed in function than the left temporo-sphenoidal lobe and the contiguous cortex in right-handed individuals. Again, the immediate rise in pulse-rate following the operation and a lessening of the high intracranial pressure illustrate the effectiveness of the decompressions and also the avoidance of the dangerous medullary compression of extreme degree. It is rather surprising that the fracture of the skull did not enter the nose, mouth or ears; if it had there would undoubtedly have been a definite lessening of the intracranial pressure by means of the escape of blood and cerebrospinal fluid through these channels of exit. It is remarkable that this patient has not been impaired mentally and particularly emotionally, and patients of this character of excellent recovery should encourage us to avail ourselves of all means and not to lessen or delay our efforts in trying to obtain good results.

In the treatment of shock following cranial injuries, there is possibly no one measure so effective and valuable as external warmth and heat applied to the entire body by means of heated blankets, hot-water bottles and hot rectal enemata and continuous warm irrigation. This patient was considered moribund upon admission—the respirations being so shallow and imperceptible that only 6 per minute were registered; on the contrary, the pulse was 144, whereas the temperature was only 97.4 and the blood-pressure 104—signs of extreme shock, and the patient should be considered only from the standpoint of the condition of shock and so treated; if survival occurs, then the associated cranial or bodily condition can be ascertained and treated accordingly.

It is unfortunate in this patient, too, that a lumbar puncture with a removal of cerebrospinal fluid was not performed just before the dura was opened at the first operation; this procedure would possibly have prevented the extensive laceration of the underlying cortex by lowering the intradural pressure, and the danger of performing a lumbar puncture at this time—the dura being opened—is practically nil, there being little or no danger of the medulla being forced down into the foramen magnum in supratentorial lesions.

ACUTE SEVERE BRAIN INJURIES ASSOCIATED WITH EXTREME SHOCK AND NO INCREASE OF THE INTRACRANIAL PRESSURE; NATURALLY, NO OPERATION. DEATH; AUTOPSY.

If the immediate shock of the cranial injury is severe, then the general blood-pressure is low—even 100 and below, so that it would be very difficult and practically impossible for a large intracranial hemorrhage to occur—even in the presence of a large intracranial vessel being torn (usually a sinus or cortical vein) owing to this lowered blood-pressure of shock; that is, the intracranial pressure would quickly become higher than this lowered blood-pressure of shock, so that no bleeding intracranially could then occur, and no large hemorrhage could result until the general blood-pressure is raised by the survival of the patient from this extreme condition of shock. Fortunately, this recovery from the shock results in many patients, and by the time it does occur the torn intracranial vessel or vessels have become thrombosed, so that, as the general blood-pressure does ascend upon the disappearance of the shock, yet very little intracranial hemorrhage results—apparently Nature's method of protecting the organism.

These patients, therefore, in the extreme condition of initial shock rarely disclose the signs of an increased intracranial pressure and therefore no cranial operation of decompression is indicated—no matter how badly the skull may be fractured nor how certain approaching death may seem; a cranial operation in this period of severe shock takes away the chance of the patient to recover from the shock; and if the patient should recover, however, from the operation, then he does so in spite of the operation.

In rare cases, where the shock immediately following the injury was slight and yet a large intracranial vessel was torn, then the intracranial hemorrhage may be so large that its rapid formation may precipitate an extreme condition of shock so that within a period of thirty minutes or an hour and before the patient reaches the hospital, the condition may be one of such extreme shock even in the presence of a high intracranial pressure that a cranial operation to relieve the increased pressure would be merely an added shock and the death of the patient would thus only be hastened. During the past five years, I have operated upon a number of these moribund patients (eight patients in all)—in extreme shock and in the presence of high increased intracranial pressure, and the percentage of recovery was 25 per cent. (two patients); I now feel that these patients would have recovered from the extreme condition of shock without an operation and then later the operation could have been more safely performed—that is, they recovered in spite of the operation; also, of the six patients who died, possibly two of them and even three might have recovered from the severe condition of shock if an operation had not been attempted at the time it was performed, and therefore the operation may have been an important factor in their deaths.

In conclusion, it can be stated that no patient in severe shock from a cranial injury should be operated upon—no matter how high the intracranial pressure is or seems to be, and that all therapeutic measures should be directed toward the recovery of the patient from the shock; if this can be accomplished, then the cranial operation of decompression and drainage can be safely performed. Naturally, no patient, whether in severe shock or not, who does not exhibit the signs of a high intracranial pressure should be operated upon—and especially a so-called "decompression" performed, as this operation presupposes the presence of an increased intracranial pressure and it surely cannot be a decompression if there is no increased pressure present. Many of the catastrophies of cranial surgery upon these patients have resulted from the neglect and oversight of this cardinal principle.

If it is possible for the patient to recover from this severe condition of shock, then it will be observed that the subnormal temperature, lowered blood-pressure and increased pulse- and respiration-rates will gradually change until the temperature becomes normal and usually above normal, the blood-pressure rises and the pulse- and respiration-rates decrease to 110 and 24 and below, respectively; now, and not until this time, is the ideal period for careful neurological examinations to be made and the intracranial condition ascertained as accurately as possible so that it will be known later whether the condition of the patient is really improving or not, and thus the appropriate treatment can be early instituted.

Acute severe brain injuries with no increase of the intracranial pressure; no operation. Early death due to extreme shock. Autopsy.

A. *Fracture of the skull present.*

Case 58.—Acute severe brain injury with no signs of an increased intracranial pressure and in severe shock. No operation. Death. Autopsy.

No. 97.—Hugh. Forty-four years. White. Married. Waiter. U. S. Admitted February 7, 1914, Polyclinic Hospital. Referred by Doctor Alexander Lyle.

Died February 7, 1914—2 hours after admission.

Family history negative.

Personal history negative.

Present Illness.—While crossing the street, patient was struck by a taxicab; immediate loss of consciousness; brought to the hospital in the taxicab.

Examination upon admission (15 minutes after injury).—Temperature, 98°; pulse, 110; respiration, 30; blood-pressure, 114. Profound unconsciousness and in a severe condition of shock: cold, clammy skin with cold perspiration over the entire body; pulse scarcely perceptible, while respirations very shallow. Laceration of scalp over left parietal area. Slight bleeding from nose but not from mouth or ears; much mastoid ecchymosis. Severe injury to chest with fracture dislocation of sternum and subcutaneous emphysema over entire chest and in both axillæ. Pupils dilated, left possibly larger than right. Reflexes all abolished; corneal reflexes absent. Fundi negative.

Treatment.—Vigorous shock measures instituted, especially external heat to entire body by means of warm blankets and hot-water bags, hot enemata of black coffee, brandy subcutaneously with atropine and strychnia hypodermically. Edema of the lungs appeared, however, within one hour and patient progressively became worse in that the temperature descended to 97.2°, pulse ascended to 150 plus and respirations to 40 plus, while the blood-pressure fell to 80 and below. Patient died 2 hours after admission from shock.

Autopsy.—Linear fracture of the vault of 4 inches in length extended from posterior portion of left parietal bone forward and obliquely downward toward left external orbital process, but it did not reach the base (Fig. 83). No extradural hemorrhage. Cerebrospinal fluid blood-tinged but no intracranial hemorrhage nor cerebral laceration could be ascertained—merely a rather pale and anemic brain with much cerebrospinal fluid at the base. Ventricles negative.

Remarks.—No treatment whatever apparently could have caused the recovery of this patient; these severe chest injuries associated with the cranial injury produced a degree of shock which could not be survived. The clinical history of subnormal temperature and initial low blood-pressure, while the pulse and respration were never below 110 and 30, respectively, indicate the condition of true shock and for these patients naturally no operation of any severity, cranial or otherwise, can be considered, as operations at this stage are merely an added shock; if, however, the shock could be survived as indicated by an increase in temperature and blood-pressure and the lowering of the pulse- and respiration-rates, and, in addition, if the signs of an increased intracranial pressure of marked degree appear, then a cranial operation for the relief of the increased intracranial pressure may be considered—such as the subtemporal decompression.

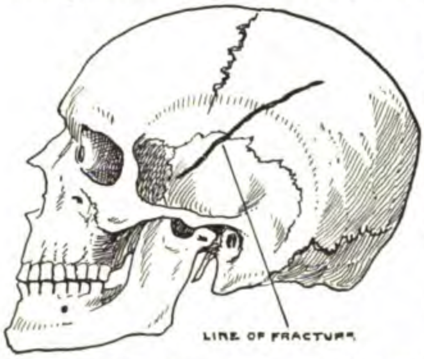


FIG. 83.—Wide linear fracture of left vault in a patient who died from shock; there was no increase naturally of the intracranial pressure and no intracranial hemorrhage was ascertained at autopsy.

As the condition of severe shock presupposes a low blood-pressure, it is only in very rare cases of cranial injuries that the signs of a marked increase of the intracranial pressure are to be ascertained during this stage of severe shock; when the signs of intracranial pressure do appear in these patients, it indicates the lessening of the shock which permits the blood-pressure to rise and therefore the blood can be forced intracranially through any of the torn intracranial vessels. It is only when a large intracranial vessel is suddenly torn as the result of a head injury that a high increase of the intracranial pressure can occur early, because these cases of head injury are all associated with a high degree of shock. If this patient had survived the shock, it is very probable that a larger intracranial hemorrhage would have occurred than was found at autopsy.

The relative unimportance of the fracture of the skull in this patient is clearly demonstrated in that there was no underlying hemorrhage or cerebral contusion, and it was just the same as if no fracture of the skull was present. If this fracture, however, had extended into the left middle ear so that the intracranial hemorrhage and the cerebrospinal fluid could have escaped through the left ear, then, if this patient had survived the condition of shock, this line of fracture would have been possibly a great benefit to the patient in permitting by means of this drainage a lessening of the increased intracranial pressure and thus avoiding the necessity of a cranial decompression. The fracture in this patient, however, was neither a benefit nor a harm to the patient.

Case 59.—Acute severe brain injury with no signs of high intracranial pressure and in severe shock. No operation. Death. Autopsy.

No. 148.—Ella. Twenty-three years. White. Single. Stenographer. U. S.

Admitted July 8, 1914, Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Died July 8, 1914—60 minutes after admission and 80 minutes after injury.

Family history negative.

Personal history negative.

Present Illness.—While alighting from a street car, patient was knocked down by an automobile; immediate loss of consciousness; brought to the hospital in the automobile.

Examination upon admission (20 minutes after injury).—Temperature, 97.4°; pulse, 140; respiration, 36; blood-pressure, 90. Semiconscious and very restless; severe shock. Compound comminuted fracture of left tibia and left fibula. No laceration or contusion of the entire scalp. Profuse bleeding from nose and both ears; both mastoid areas ecchymotic and both orbits swollen and ecchymosed. A discharge of cerebrospinal fluid from the nose and ears observed. Urine obtained by catheter negative. Pupils dilated but equal and react sluggishly to light. Reflexes—patellar increased, right more than left; no ankle clonus, but right Babinski; abdominal reflexes absent. Fundi—retinal veins slightly enlarged; nasal margins of both optic disks not as clear and distinct as temporal margins. Lumbar puncture—blood-tinged cerebrospinal fluid under normal pressure (approximately 7 mm.).

Treatment.—Vigorous shock measures instituted, but patient became progressively worse in that temperature descended to 97°, pulse became more and more rapid until it was imperceptible and the respiration could not be counted, while the blood-pressure rapidly lessened until death occurred from the shock one hour after admission.

Autopsy.—Fracture of base extended through middle fossa and both petrous bones into the tympanic cavities, and upward on the left side of the vault to the parietal crest. Both orbital plates of sphenoid bone also fractured (Fig. 84). Both tympanic membranes lacerated in their posterior halves. No extradural hemorrhage. Tip of left temporo-sphenoidal lobe and anterior surface of both frontal lobes contused and lacerated. Cerebrospinal fluid blood-tinged and an occasional punctate hemorrhage throughout the cortex of both hemispheres, but no extensive hemorrhage intracranially could be found. Ventricles negative.

Remarks.—It was remarkable that there were no external evidences upon the head of the area of contact, and yet the fracture through the base was most extensive; whatever bleeding occurred intracranially must have escaped through the fracture into the ears, although in the presence of such a marked degree of shock, very little bleeding could have occurred intracranially. The increased reflexes, particularly upon the right side, and especially the right Babinski, were undoubtedly due to the greater cortical injury over the left hemisphere. In the presence of cortical contusions and lacerations as in this patient, if the shock could have been survived and thus the blood-pressure be increased, undoubtedly there would have been an extensive subdural hemorrhage which would become greater as the blood-pressure became higher and therefore the signs of an increased intracranial pressure would have become more and more evident. It is upon these moribund patients in shock that, formerly, cranial operations were frequently performed, because it seemed, and correctly so, that the patient was going to die, and yet any cranial operation upon these patients takes away whatever chance the patient has of surviving the shock, and if they should survive both the shock and the added burden of the operation, then they recover merely in spite

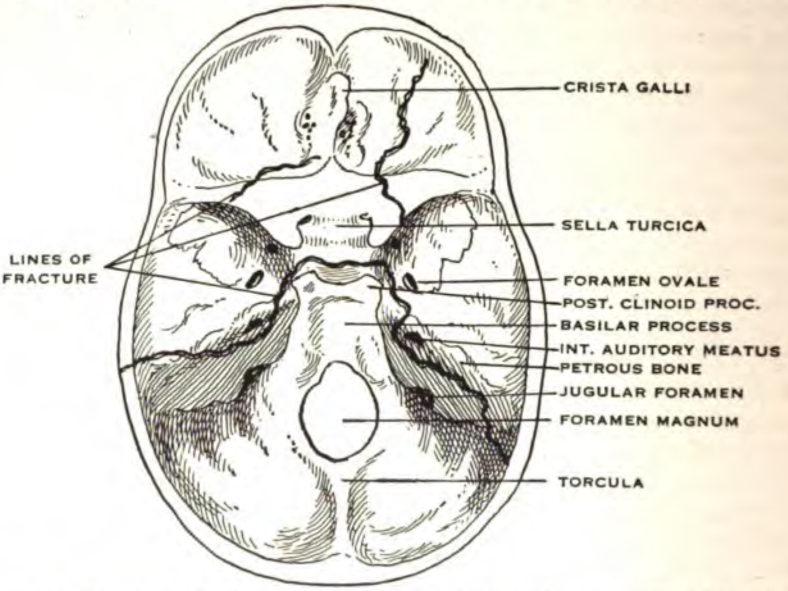


FIG. 84.—Extensive basilar fractures of the middle and anterior fossæ in a patient dying of extreme initial shock. No operation was performed as there could not be a high intracranial pressure in the presence of severe shock; any operation would have been merely an added shock to the patient.

of the operation, and naturally they would have done so—and much n easily, if the operation had not been performed.

Patients of this character in the extreme condition of shock req the most urgent treatment for the shock; as external warmth is possibly most important factor in the treatment of these shock patients, it would s advisable and practicable that ambulances should be equipped both hot water-bags and heated blankets so that these patients could rec appropriate treatment almost immediately. (A small electric heater in ambulance would be sufficient for the blankets.) It is the transfer of the patient from the street, sidewalk, or the floor, and usually "chi through," and then the ride to the hospital, especially in cold weat the cursory examination in the accident-room—it is these delays and posures before the patient reaches the ward and appropriate treatment lessen the patient's chances of recovery, and especially these patients ha severe shock.

It might be argued that patients of this type would have had a chanc recover if the operation of cranial decompression had been performed du the first hour or two after admission—because the patient died without operation and therefore he might have lived if an operation had been formed. This argument can no longer be used in that we now know tha cranial operation should be advised in these acute cases, unless in the pres of a marked increase of the intracranial pressure (excluding naturally cases of depressed fractures of the vault); this patient at no time sho the signs of an increased intracranial pressure and therefore no cra operation, and particularly a decompression operation, was indicated. escape of blood and cerebrospinal fluid from the nose possibly lessened increase of the intracranial pressure that might have been present.

The initial shock of this patient was apparently not so severe as in m patients who have survived, and for this reason it was believed that patient would be able to recover from the shock. If the shock had been vived and therefore an increase of the blood-pressure had occurred, autopsy findings indicated that undoubtedly the intracranial pressure w have been markedly increased so that the operation of cranial decompres might have later been necessary. The absence, however, in this patient large intracranial hemorrhage is further evidence that the condition of s prevents a large amount of intracranial bleeding which only occurs if shock can be survived. When patients are in a severe condition of shock, neurological examination should be a most cursory one, as the entire atten should be directed toward the treatment of shock, and prolonged exam tions cause the patient to be exposed, body heat is lost which is most t avoided and the patient is certainly not benefited as nothing really ca done for the patient until the shock has subsided—brain injury or The lumbar puncture, therefore, upon this patient was inadvisable du this period of shock and it should not have been performed; the observat however, that the pressure of the cerebrospinal fluid was only 7 mm. firms the opinion that in severe shock it is most rare for a high intracra pressure to be present and, upon a physiological basis, practically imposs

The profuse discharge of cerebrospinal fluid through the line of fracture, as revealed at autopsy, in the cribriform portion of the ethmoid bone would have been an excellent channel through which an increased intracranial pressure could have been lessened, so that the patient would thus have "decompressed" himself; the danger of infection, however, through this same line of fracture into the nasal cavity would have been very great indeed—possibly greater than in similar fractures through the middle ear.

B. *No fracture of the skull present.*

CASE 60.—Acute severe brain injury with no signs of an increased intracranial pressure but in severe shock. No operation. Death. Autopsy.

No. 100.—Arthur. Twenty-eight years. White. Single. Butler. England.

Admitted January 9, 1914, Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Died January 9, 1914—1 hour after admission and 95 minutes after injury.

Family history negative.

Personal history negative.

Present Illness.—While cleaning a fourth-story window, patient fell to the pavement below; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (35 minutes after injury).—Temperature, 97.2°; pulse, 150 plus; respiration, 40; blood-pressure, 82. Profound unconsciousness and in extreme shock; moribund; pulse imperceptible at times. Contusion over left eye; left orbit ecchymosed. No bleeding from nose, mouth or ears; no mastoid ecchymoses. Fracture of pelvis—comminuted left ilium. Pupils widely dilated and do not react. Reflexes all absent, except for slight cerebral reflex. Fundi negative. Abdominal examination negative; catheterized urine negative.

Treatment.—Vigorous shock measures instituted. Patient rapidly became worse and died in a condition of extreme shock one hour after admission.

The coroner's physician (Doctor Lehane) viewed the body at a distance of 10 feet, and, with the aid of the history, made the diagnosis of "fracture of the skull" and refused to perform an autopsy—"because the cause of death is evident." Permission, however, for the autopsy was later obtained from the relatives and it was accordingly performed.

Autopsy.—No fracture of the skull could be found; no extradural hemorrhage. Blood-tinged cerebrospinal fluid with contusion of the anterior surface of both temporo-sphenoidal lobes and the inferior surface of the left frontal lobe; otherwise the brain was negative. Very little cerebrospinal fluid in the cranial cavity. Ventricles negative.

Remarks.—This patient undoubtedly died from shock not only from his general bodily injuries, particularly the fracture of the pelvis, but also the severe head injury which had caused the contusion of areas of the cerebral cortex; no large intracranial hemorrhage was present, however, because the lowered blood-pressure of shock would not permit a large amount of intracranial bleeding in that the intracranial pressure was higher than this lowered blood-pressure of shock.

The illogical and mediæval diagnosis of "fracture of the skull" as being the cause of death, or even being an important factor in the causation of death (if we except those patients dying from a meningitis through infection by way of the fracture itself, or those patients having large depressed fractures of the skull), can no longer be used as a satisfactory explanation of the cause of death of patients following a cranial injury; the fractures of bones of the extremities and of the trunk may be and are important in the treatment of these conditions, but it does not necessarily follow that in brain injuries the fracture of the skull is of any great importance—whether it is present or not. Considering brain injuries as being necessarily associated with fractures of the skull and thus indicating the method of their treatment—this conception has retarded the progress of the treatment of brain injuries possibly more than any other one factor, and the sooner we can consider brain injuries independently of the presence of a fracture of the skull, and more in relation to the presence or not of an increased intracranial pressure, just so much earlier will these patients obtain a more rational treatment and therefore the mortality be greatly lowered.

Case 61.—Acute severe brain injury with no signs of increased intracranial pressure and in an extreme condition of shock. No operation. Death. Autopsy.

No. 106.—John. Thirty-five years. White. Married. Storekeeper. U. S.

Admitted January 12, 1914, Polyclinic Hospital. Referred by Doctor John A. Bodine.

Died January 12, 1914—30 minutes after admission and 55 minutes after injury.

Family history negative.

Personal history negative.

Present Illness.—Patient fell from a platform in the subway and was struck by a train; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (25 minutes after injury).—Temperature, 97.2°; pulse, 118; respiration, 32; blood-pressure, 106. Profound unconsciousness, but became conscious for 15 minutes sufficiently to realize his condition and then became again unconscious; in severe shock. Stellate lacerations of the scalp over left frontal area and above left eye; left orbital ecchymosis. No bleeding from nose, mouth or ears; no mastoid ecchymoses. Pupils widely dilated and do not react to light. Reflexes all absent, including corneal reflexes. Fundi negative.

Treatment.—Vigorous shock measures instituted. Patient, however, became progressively worse—the pulse- and respiration-rates ascending to 150 plus and 50 plus, respectively, until imperceptible, while the blood-pressure sank lower and lower until it could not be registered; death occurred 30 minutes after admission. (The ambulance surgeon stated that at the time the patient was carried into the ambulance the pulse was 66 while the respiration was only 8—that is, the stage of acute medullary compression; this observation could not be confirmed, however, by the autopsy findings.)

Autopsy.—(Permission was received from relatives after the coroner's physician, Doctor Weston, had refused to perform an autopsy since "the diagnosis is evident from the history," and, "the scalp wounds and the black [illegible] a fracture of the skull." No fracture of the skull ascertained; no extradural hemorrhage. Brain itself very "wet" and edematous but no cortical hemorrhages or lacerations could be found. Slightly blood-tinged cerebrospinal fluid, especially in the middle fossa. Medulla negative. Ventricles negative. Subtentorial fossa negative.

Remarks.—The initial shock of this patient was sufficient in itself apparently to cause death, and yet the autopsy findings hardly account for the death—especially in a man apparently in good health under middle age. If this patient could have received the ideal shock treatment immediately after the injury instead of lying upon the cement floor of a subway station for a period of 20 minutes, it might have been possible to have obtained a recovery; there was no evidence of chronic alcoholism, although the man was a rather heavy-set, obese patient, yet ordinarily the shock might have been survived. It was unfortunate that permission for a complete autopsy of the body was not obtained, for possibly there were other internal injuries of the abdomen or chest that would have explained the death of this patient.

The absence of definite signs of pressure about or in the medulla and subtentorially in the autopsy findings is rather puzzling in view of the observation of the ambulance surgeon that the patient upon admission to the ambulance had a pulse-rate of 66 and a respiration-rate of only 8—the typical rates of pulse- and respiration-rates in acute medullary compression; the medulla, however, was of normal appearance and consistency so that it is very difficult to explain this observation as being due to a medullary disturbance. The blood-pressure would have been an interesting observation at this period; if high or even slightly increased, it would tend to confirm a diagnosis of medullary compression, whereas if low then the condition of shock and other extracranial lesions might be considered; in either case, however, it is very difficult to explain, and it is very unfortunate that permission for a complete autopsy was not obtained.

Acute Severe Brain Injuries Associated with an Extreme Intracranial Pressure Precipitating an Acute Medullary Edema. No Operation. Death: Autopsy.

The acute medullary edema occurring in these patients following a cranial injury with and without a fracture of the skull is due to an extreme intracranial pressure, either of hemorrhage or of an excess amount of cerebrospinal fluid (cerebral edema). This increase of intracranial pressure may occur so rapidly following the cranial injury that the symptoms and signs of shock merge quickly into those of medullary edema—at times apparently without passing through the clinical stages of medullary compression—that is, the subnormal temperature, low blood-pressure and increased pulse- and respiration-rates of severe shock quickly become changed, as the result of a very early and extreme intracranial pressure, to a high temperature, and even higher pulse- and respiration-rates of medullary edema without showing clinically the signs of a preceding medullary compression—slightly increased temperature, an ascending blood-pressure and descending pulse- and respiration-rates to 60 and 16, respectively, and lower. Formerly, many of these patients succumbing to an early medullary edema were believed to have died from the condition of extreme shock as the more modern methods of determining this increased intracranial pressure by means of the ophthalmoscope and the spinal mercurial manometer were not in common use, and therefore the true intracranial condition was overlooked; these cases can now be easily differentiated clinically, and if an autopsy is performed (and permission for it should always be obtained), the diagnosis of medullary edema resulting from the high intracranial pressure can thus be confirmed.

In the patients who recover from this shock of varying degree and then exhibit the signs of an increasing and high intracranial pressure, unless this increased intracranial pressure—it matters not whether due to hemorrhage or edema—is relieved early and thus the signs of medullary compression are lessened, then the great danger of a medullary edema is to be feared; for if once the lowered pulse- and respiration-rates and increased blood-pressure of medullary compression quickly change to the high and increasing pulse- and respiration-rates and descending blood-pressure of medullary edema, then that patient will die—operation or no operation—within 24–36 hours. It is of the greatest importance, therefore, that these patients should be carefully and repeatedly examined in order to estimate accurately the true intracranial status and especially in regard to an increasing intracranial pressure; if the expectant palliative method of treatment does not prevent the intracranial pressure from increasing, then its early lowering by means of a subtemporal decompression and drainage, and if necessary, a bilateral decompression is advisable.

Acute severe brain injuries associated with high intracranial pressure precipitating medullary edema. No operation. Death. Autopsy.

A. *Fracture of the skull present.*

a. High intracranial pressure due to a large intracranial hemorrhage.

Case 62.—Acute severe brain injury associated with high intracranial pressure due to subdural hemorrhage and cerebral edema. No operation. Medullary edema; death. Autopsy.

No. 140.—James. Fifty-five years. Married. Horse-shoer. Ireland.

Admitted April 2, 1914, Polyclinic Hospital. Referred by Doctor W. S. Bainbridge.

Died April 2, 1914—9 hours after admission and 10 hours after injury.

Family history negative.

Personal history negative.

Present Illness.—Patient was found lying upon the sidewalk; unconscious; brought to the hospital in the ambulance.

Examination upon admission (at least one hour after injury).—Temperature, 102.6°; pulse, 90; respiration, 28; blood-pressure, 140. Profound unconsciousness; odor of alcohol upon breath—giving the house surgeon the impression that patient was merely alcoholic. Small hematoma of contact over left occipital area. No bleeding from nose, mouth or ears. Slight spasticity of left arm and left leg. Pupils—right dilated while left pupil very much contracted and no reaction to light. Reflexes: patellar—left greater than right; suggestive left Babinski but no ankle clonus; abdominal reflexes absent. Fundi—retinal veins dilated; nasal halves of both optic disks slightly blurred, but as there was a profuse congestion of both retinæ, it was at the time considered as being due to chronic alcoholism. No lumbar puncture performed as it was considered unnecessary (an unfortunate mistake).

Treatment.—Expectant palliative.

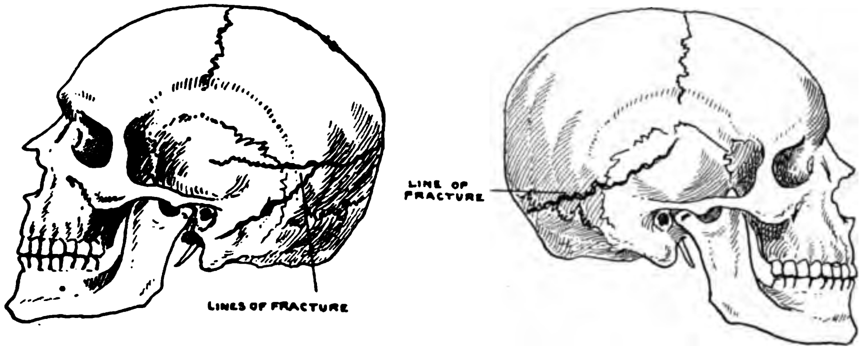
Examination (4 hours after admission).—Temperature, 103°; pulse, 60; respiration, 16; blood-pressure, 140. General condition of the patient practically the same except that the pulse- and respiration-rates were descending and the signs of intracranial pressure as registered upon the fundi were increasing. Definite left mastoid ecchymosis. Pupils—both dilated and do not react to light. Reflexes: patellar—both exaggerated, right possibly more than left; double Babinski and exhaustible ankle clonus; abdominal reflexes absent. Fundi—retinal veins dilated; nasal halves of both optic disks blurred and the temporal margins were obscured by edema. Lumbar puncture—bloody cerebrospinal fluid under increased pressure (approximately 17 mm.).

Treatment.—The expectant palliative treatment was continued in the hope and belief that the patient himself would be able "to take care of" this increased pressure and thus an operation be avoided. (The operation, however, should have been performed at this time while the pulse was descending and before the lowest pulse-rate of medullary compression had occurred.) The patient continued in practically the same condition for two hours when he suddenly became worse, in that the temperature became 103.6°; pulse, 100; respiration, 24; blood-pressure 130—that is, the pulse-rate ascending rapidly while the blood-pressure was descending—the typical signs of medullary edema. The patient was hurried to the operating room in the mistaken belief that an immediate subtemporal decompression might cause a recovery of life, but the pulse ascended so rapidly to 150 plus while the blood-pressure descended to below 100 that the patient died before the operation could be begun.

Autopsy (Doctor C. A. Schultze).—Large hematoma evenly distributed over entire right temporo-parietal area. Line of fracture began in the left occipital area (underlying the point of contact) and extended horizontally around the skull—on the left side to the left external orbital process and on the right side to a point one inch above the right external auditory canal (Figs. 85 and 86). Small hemorrhage beneath the torn periosteum of right orbital plate, but orbital bone itself intact. Middle ears negative. No epidural clot. Dura was intact—even under the line of fracture. Very large subdural clot over entire right hemisphere—about 16 ounces in amount and almost the size of a small grapefruit. Numerous supracortical clots and cortical lacerations of both anterior frontal lobes and also the tips of both temporo-sphenoidal lobes—more on the right side; extensive lacerations of right temporal lobe. No subtentorial hemorrhage. Ventricles negative.

Remarks.—If this patient could have been treated early by means of a right subtemporal decompression and while the pulse-rate was descending, he might have been given a definite chance of recovery. The autopsy findings are most instructive in that such a huge subdural hemorrhage of almost 4 inches in diameter could be present in the right parieto-temporal area, and yet the only localizing signs of its presence, beside the signs of increased intracranial pressure which were not marked nor extreme, were the slight spasticity of the left arm and left leg and the increased reflexes of the left side and a suggestive left Babinski at first, while the left pupil was dilated in the beginning. These findings merely illustrate in a small way the great difficulty of localizing accurately small acute lesions of the brain when not situated in the motor cortex, and also to emphasize again that it is not so important to localize the lesion from an operative standpoint if an early subtemporal decompression is only performed upon the side of the hemisphere over which the greater hemorrhage has occurred, and also that, in the usual case, it is more a question of lowering the increased intracranial pressure whether due to hemorrhage or cerebral edema; naturally in this patient, who is, I believe, an exception, it would have been most important to have drained the hemorrhage itself, and undoubtedly that would have been done if the right subtemporal decompression had been performed as planned.

This case also emphasizes the importance of examining these patients early and frequently by the most accurate methods now known of determining an increased intracranial pressure by considering the entire clinical picture of pulse- and respiration-rates, blood-pressure, pupillary inequality and—most important—repeated ophthalmoscopic examinations of the fundi and the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer. Also to put us upon our guard that merely because a patient has the odor of alcohol upon his breath—this fact should not make us feel that the patient is unconscious because drunk, but most careful examinations should be made and the fact borne in mind that if there is any cranial lesion and if the patient in addition is drunk, then in the presence of an increaesd intracranial pressure not only does the alcoholism tend to mask the symptoms and signs of the intracranial lesion but to render the patient much more susceptible to the onset of an acute medullary edema; and therefore this great danger should always be most carefully considered. If a lumbar puncture had been performed at the time of this patient's admittance to the hospital, and this is usually done unless the patient is in shock, and this patient was *not* in shock, then the presence of blood would have been ascertained in the cerebrospinal fluid



FIGS. 85 AND 86.—Tremendous linear fracture extending horizontally around the posterior half of the vault in a patient developing an acute and early edema of the medulla, due to an extreme intracranial pressure. A decompression operation performed during the stage of medullary compression might have offered this patient a chance of recovery.

and this patient would naturally have received much more careful treatment in that the gravity of the condition would have been realized early; also, if an early X-ray picture had been taken, the line of fracture would undoubtedly have been revealed so that the factor of alcoholism would not have appeared to be such an important one in the case; it is very probable that the true condition of this patient would have been early recognized and especially by means of the lumbar puncture with a measurement of the pressure of the cerebrospinal fluid, so that a right suptemporal decompression might have been sufficient to have obtained an excellent recovery.

Case 63.—Acute severe brain injury associated with extreme intracranial pressure due to subdural hemorrhage and cerebral edema. No operation. Medullary edema; death. Autopsy.

No. 138.—Lorenzo. Forty years. White. Married. Ironworker. Italy.

Admitted April 7, 1914, Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Died April 7, 1914—7 hours after admission and 8 hours after injury.

Family history negative.

Personal history negative.

Present Illness.—Patient was found lying upon the sidewalk; unconscious; brought to the hospital in the ambulance.

Examination upon admission (at least an hour after injury).—Temperature, 102.4°; pulse, 150; respiration, 32; blood-pressure, 112. Profound unconsciousness with the patient in a moribund condition of medullary edema. Well-developed robust man; chronic alcoholism. Small hematoma over left occipital region with extensive left mastoid ecchymosis. Profuse bleeding from nose. Occasional twitchings of both arms which are slightly spastic. Respiration irregular but shallow. Pupils dilated, equal and do not react to light. Reflexes all abolished, except corneal reflex. Fundi—retinal veins dilated; nasal halves of both optic disks obscured by edema. Lumbar puncture—bloody cerebrospinal fluid under high pressure—accidentally spurted a distance of 2 feet (approximately 24 mm.). Urine—large trace of albumen with many hyaline and granular casts and a slight trace of sugar.

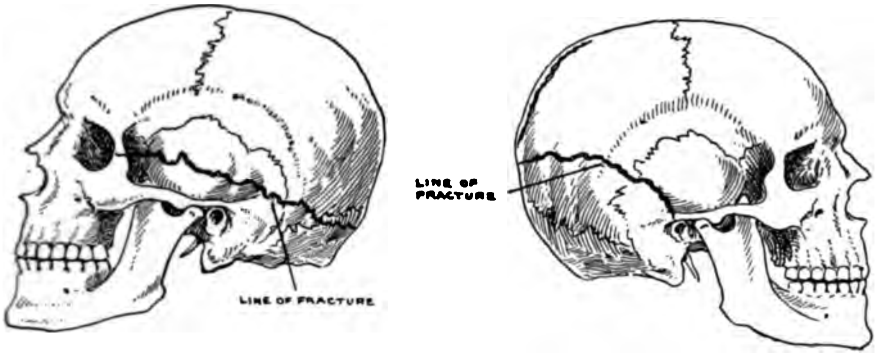
Treatment.—Expectant palliative. The condition was considered one of acute medullary edema, but patient was carefully "watched" in the forlorn hope that his condition might be improved. Within one hour after admission, both fundi revealed double "choked disks"—the left having a swelling of 4 diopters while the right was 3 diopters of swelling; retinal veins became tortuous and obscured by the retinal edema. Two hours after admission, a double Babinski appeared and the hematoma over left occipital area increased in size until it infiltrated the entire posterior portion of the scalp and the left side of head. The pulse gradually became imperceptible, edema of the lungs occurred and the patient died 7 hours after admission.

Autopsy (Doctor C. A. Schultze).—Large subpericranial hematoma over entire portion of left side and back of head. Main fracture extended transversely around the entire posterior portion of the skull and into both mastoid bones (Figs. 87 and 88); another line of fracture extended from the occipital protuberance forward in the median line for a distance of about 4 inches.

Dura intact with no extradural hemorrhage. Many subdural clots, especially over temporo-sphenoidal lobes, and anterior surface of both frontal lobes. Extensive lacerations of both temporo-sphenoidal lobes, especially right. Brain itself very swollen and edematous and typically "waterlogged." No subtentorial hemorrhage nor evident direct compression of the medulla. Hemorrhage in left middle ear, although tympanic membrane intact and no fracture of left petrous bone, but the overlying periosteum was torn—that is, it is possible to have a hemorrhage into the middle ear without there necessarily being a fracture of the contiguous portion of the petrous bone.

Remarks.—It would have been foolhardy to have advised an operation upon this patient because the signs of extreme medullary edema were already present, and any operation, no matter how slight, would have merely hastened the exitus. Such patients are doomed—operation or no operation.

The absence of direct medullary compression subtentorially in this patient indicates that the supratentorial pressure must have been exceedingly high to have produced the signs of medullary edema so early, as the condition of medullary edema was very much advanced, even at the time of admission of the patient.



FIGS. 87 AND 88.—Tremendously wide horizontal linear fracture of the posterior half of the vault in a patient whose extreme intracranial pressure of subdural hemorrhage and cerebral edema had precipitated an acute medullary edema at the time of his admission to the hospital. Expectant palliative treatment in the hope that a recovery might be possible; naturally, no operation.

The presence of bilateral spasticity, and particularly of both arms and legs in these patients, indicates a most serious intracranial condition of compression and edema of the pyramidal tracts in the internal capsule and in the medulla itself, and its existence usually indicates the condition of medullary edema and an early death of the patient. Each patient who has developed this condition of bilateral spasticity, with or without convulsive seizures in this series of patients, has died from an early medullary edema; this condition of spasticity frequently occurs after the onset of the medullary edema and is merely another bad prognostic sign.

The frequent laceration of either temporo-sphenoidal lobe, and especially their tips, is usually a latent condition, and in no way, I believe, complicates the prognosis providing the increased intracranial pressure due to the consequent hemorrhage and cerebral edema is relieved. The inferior and anterior portions of both *frontal* lobes are similarly affected, but not so frequently as the anterior tips of the temporo-sphenoidal lobe. It must always be remembered, however, that it is not these cerebral lacerations which cause the death of the patient, but it is the unrelieved intracranial pressure resulting from the brain injury; if this intracranial pressure is lessened to within normal physiological limits, then these lacerations of the so-called "silent" areas of the brain will take care of themselves.

It is rather unusual for the signs of increased intracranial pressure to progress so rapidly as they did in this patient during the stage of medullary edema—particularly is this true of the ophthalmoscopic findings in that "choked disks" developed within one hour after admission of the patient to the hospital and having a measurable swelling of 4 diopters. The subpericranial hematoma undoubtedly enlarged as a result of blood escaping through the underlying fracture of the vault, but this means of natural decompression was not of sufficient amount to prevent the intracranial pressure from increasing rapidly.

CASE 64.—Acute severe brain injury associated with signs of high intracranial pressure due to subdural hemorrhage and cerebral edema. No operation. Medullary edema; death. Autopsy.

No. 421.—Henry. Forty-two years. White. Married. Salesman. U. S.
Admitted October 27, 1915, Polyclinic Hospital.
Died November 17, 1915—21 days after admission and injury.

Family history negative.

Personal history negative, except for chronic alcoholism—becoming drunk at least once a month.

Present Illness.—Patient was found lying unconscious at the bottom of a stairway; brought to the hospital in the ambulance.

Examination upon admission (at least one hour after injury).—Temperature, 97.4°; pulse, 88; respiration, 24; blood-pressure, 118. Poorly nourished; unconscious and in severe shock. No bleeding from nose or mouth, but profuse hemorrhage from both ears; left mastoid ecchymosis. Pupils slightly enlarged and do not react to light. Reflexes all absent. No ophthalmoscopic examination or lumbar puncture performed on account of the severe shock.

Treatment.—Expectant palliative. The pulse gradually descended from 88 to 62, while the blood-pressure increased to 148 and the temperature to 100.6°, and they remained practically in this same ratio for the following 4 days when the pulse ascended to 92, where it remained until the consultation. Patient became conscious on the fifth day after admission and apparently improved daily. Two days ago, however, the patient became confused mentally, mildly delirious and rapidly became worse, so that acute delirium tremens was suspected.

Examination (for the first time at consultation, on November 8, 1915—12 days after admission).—Temperature, 101.8°; pulse, 90; respiration, 26; blood-pressure, 124. Poorly nourished; extremely restless and continuously talking in a mildly delirious condition. Although patient had been accustomed to the daily use of alcohol, since entering the hospital patient had not received alcohol in any form. Definite right facial paralysis of cortical type (muscles of right forehead not being involved); no weakness of right arm or of right leg. Otoscopic examination of both ears—both tympanic membranes lacerated in their posterior halves. Pupils—left dilated, both irregular and do not respond to light. Reflexes: patellar active—right possibly more active than left; no ankle clonus, but tendency to right Babinski; abdominal reflexes cannot be elicited. Fundi—retinal veins dilated; nasal halves of both optic disks obscured by edema—left more than right. Lumbar puncture—bloody cerebrospinal fluid under high intracranial pressure (approximately 19 mm.). During the examination, the patient was continuously picking at the bed-clothes (carphologia) and looking anxiously about as though in a mild terror. Diagnosis was fracture of the skull (base) with an increased intracranial pressure due to cerebral edema (predisposed by alcoholism) and a possible intracranial hemorrhage; delirium tremens.

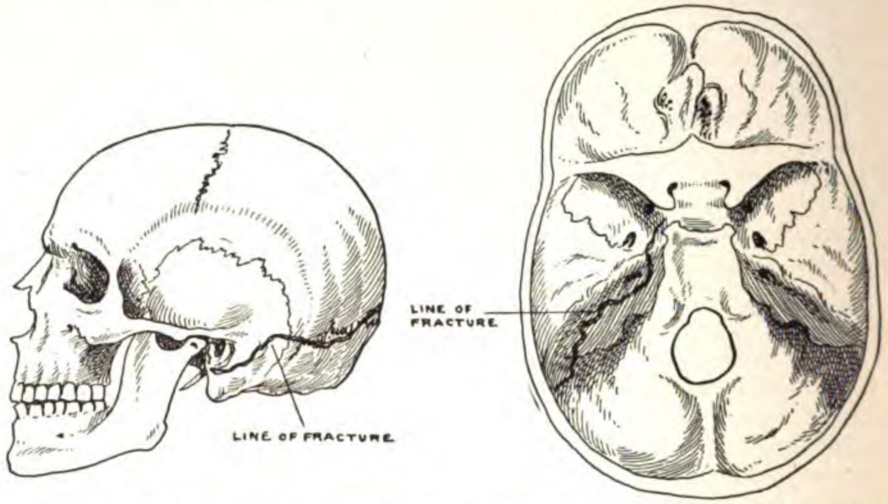
Treatment.—Owing to the complication of chronic alcoholism with the signs of a mild delirium tremens appearing, the expectant palliative treatment was considered advisable—the patient to receive small amounts of alcohol frequently and the general treatment of chronic alcoholism with its acute manifestations should be administered. In spite of this treatment, however, the patient neither improved nor became markedly worse until 9 days after consultation, when the temperature suddenly ascended to 107 plus, pulse 150 plus, respirations 40 plus, while the blood-pressure descended to below 80, so that the patient consequently died—9 days after consultation and 21 days after injury.

Autopsy.—Liver was of the typical hobnailed character. Kidneys small and white. Lungs—entire right apex filled with tubercles, which were also scattered throughout the upper portions of both lungs. Heart negative.

Cranial.—Line of fracture extended from left occipital bone forward and downward obliquely through left mastoid bone and then along the petrous portion to the sella turcica; no fracture of the right petrous bone found (Figs. 89 and 90). Left hemisphere covered by a thin layer of subdural blood while the brain itself together with the cerebellum and the medulla was very much swollen, edematous and "water-logged." Ventricles negative. No intracerebral hemorrhage or cortical laceration ascertained. Pathological diagnosis: fracture of skull, subdural hemorrhage, cerebral edema due to head injury associated with chronic alcoholism, pulmonary tuberculosis and chronic nephritis.

Remarks.—As the autopsy findings indicate, this patient apparently had little chance of recovering from a severe brain injury—operation or no operation. It was only within the first 4 days following the injury when the pulse had descended to 62 and had remained there for 4 days while the blood-pressure had risen to 148, and there being present the other signs of an increased intracranial pressure—it was only during this period that an operation of subtemporal decompression could have been considered advisable.

It would seem that the condition of chronic alcoholism had been the more important factor in this patient and that it should have been given more importance in the treatment. To patients of middle age who have been accustomed to taking a daily amount of alcohol and then larger quantities at irregularly frequent intervals, the deprivation of their daily allowance of alcohol is of great importance to their bodily functions, and the danger of withdrawing suddenly their daily allowance is so great for fear of precipitating both physical and emotional disturbances that it is of the utmost importance that these patients receive small and frequent doses of alcohol daily. Once the signs of mild delirium tremens occur in patients having had brain injuries, then it is usually too late to prevent the progress of the condition, although in this particular patient, the conditions progressed very slowly and it was not until the day of death that hope was given up of the patient's recovery. Repeated lumbar punctures might have been of great value to this patient in that the shock of such a procedure was practically nil, and after the withdrawal of cerebrospinal fluid from patients having acute "wet" brains due to chronic alcoholism and the allied toxic conditions, very frequently a marked improvement results—even if only of temporary duration; in selected patients, daily repeated lumbar punctures may be of permanent value to the patient in lessening the delirium and extreme restlessness, and even making it possible for their nourishment to be taken with little or no difficulty.



Figs. 89 and 90.—Extensive linear fracture of left occipital and the petrous portion of the left temporal bones in a patient developing an acute medullary edema upon the twelfth day following the cranial injury. Beside the cranial injury, it was ascertained at autopsy that the resistance of the patient had been lowered by the effects of chronic alcoholism, pulmonary tuberculosis and chronic nephritis.

After the first 4 days following the patient's admission to the hospital, a subtemporal decompression or any operation of major character would have, in my opinion, resulted in the earlier death of the patient, and if any operation at all was to have been performed, it should have occurred within the 4 days following the patient's admission and after the signs of initial shock had disappeared.

It was very interesting at the autopsy to elicit a fracture of the skull only of the left petrous bone and not of the right petrous bone, although the right tympanic membrane had been lacerated and a profuse discharge of blood had occurred through the opening; this observation again emphasizes the point that a mere laceration of the tympanic membrane and discharge of blood from the ear, and no cerebrospinal fluid observed, does not presuppose the existence of a fracture of the adjacent bones of the ear—it is only when cerebrospinal fluid is observed in the discharge from the ear that a fracture of the petrous bone can be said to have occurred. In this patient, the bleeding from the right ear undoubtedly resulted from the laceration of the right tympanic membrane itself, but it is difficult to conceive of the right tympanic membrane being lacerated without a fracture of the adjacent bones having occurred and yet no such fracture could be ascertained by most careful examination. Bleeding into the middle ear can occur without a fracture being present and the tympanic membrane intact, similar to orbital and subconjunctival ecchymoses and no fracture of the orbital bones being present; there is frequently, however, a tear of the surrounding periosteum or of the dura over the bones in these patients.

This is another case of direct medullary compression resulting in an early medullary edema which occurred so rapidly and before the signs of shock had disappeared that it was impossible to perform an operation for the relief of the pressure. It has been my experience that if these early patients with head injuries have a lowered temperature and blood-pressure, and yet the pulse is also lowered, then the condition of direct medullary compression should be considered as the factor complicating the initial shock following the injury. Naturally, it would be unwise during this period of shock to perform any operation to relieve medullary compression because the death of the patient would result from the added shock of the operation itself; and if this condition of shock merges directly into the condition of medullary edema without passing clinically through the stage of medullary compression, then also is an operation not to be considered because it would be of no value in retarding the progress of medullary edema —in fact, it would merely hasten it. But if the patient recovers from the condition of shock so that the temperature rises to normal and above, and the blood-pressure ascends to over 120, and yet the pulse remains low and there are present the other signs of an increased intracranial pressure as revealed by the ophthalmoscope and the spinal mercurial manometer, then this is the stage for the operation of decompression to be performed; that is, these patients must be most carefully watched and frequently examined— the pulse, respiration and blood-pressure being taken every 30 minutes, and as soon as the shock disappears, then the use of the spinal mercurial manometer at lumbar puncture and the frequent examination of the fundi with the ophthalmoscope.

The autopsy findings are interesting in that the early contraction to pin-point of the left pupil and the paresis of the right arm and right face were due to the subdural hemorrhage overlying the left cortex, and as the hemorrhage increased in size beyond the irritative stage, the left pupil became dilated from the paralytic effect of a large supracortical and ipsolateral hemorrhage; a complete paralysis of the right side of the body was not demonstrated.

It is fortunate that a lumbar puncture had not removed much of the spinal fluid at the first examination, as it might have been considered a factor in precipitating the acute medullary edema, especially in view of the autopsy findings in the subtentorial fossa; the repeated lumbar punctures were performed after the medullary edema had become advanced.

b. High intracranial pressure due to cerebral edema alone, and no intracranial hemorrhage present.

CASE 65.—Acute severe brain injury associated with high intracranial pressure due to cerebral edema. No operation. Medullary edema; death. Autopsy.

No. 533.—Edward. Twenty-two years. White. Single. Clerk. U. S. Admitted March 21, 1916, Polyclinic Hospital.

Died March 21, 1916—3 hours after admission and 3½ hours after injury.

Family history negative.

Personal history negative.

Present Illness.—While delivering a package at the side door of a house, patient tripped over a chain, falling backward to the ground upon his head; no loss of consciousness but complained of severe pain throughout the head; brought to the hospital in the delivery wagon.

Examination upon admission (35 minutes after injury).—Temperature, 98°; pulse, 38; respiration, 14; blood-pressure, 132. Semiconscious, but became unconscious within 30 minutes after admission. No bleeding from nose, mouth or ears; no mastoid ecchymoses. Pupils equal, moderately contracted and reacted to light sluggishly. Reflexes—patellar moderately increased but equal; no ankle clonus nor Babinski; abdominal reflexes were equal but depressed (within one-half hour they could not be elicited). Fundi—retinal veins slightly enlarged; both optic disks clear and distinct—there being no signs of an increased intracranial pressure. Lumbar puncture—slightly blood-tinged cerebrospinal fluid under high intracranial pressure (approximately 18 mm.); (the blood was apparently a contamination of the puncture as it was not mixed evenly throughout the cerebrospinal fluid).

Treatment.—Owing to the signs of extreme intracranial pressure as registered by the very slow pulse- and respiration-rate and also at the lumbar puncture, preparations for an immediate operation were made in the hope that a medullary edema would not be precipitated by these signs of high medullary compression. In the meantime, the patient was admitted to the ward, head prepared for operation, 6 ounces of hot black coffee administered per rectum, camphor in oil (grains 5), atropine (grains $\frac{1}{50}$) hypodermically, and hot water-bags and heated blankets applied to body; 16 ounces of urine were removed by catheter. The condition of the patient, however, rapidly became worse; the temperature descended to 97° (rectal), the pulse ascended to 48 within an hour and to 72 within an hour and a half, and to 140 within 2 hours after admission, while the respiration increased correspondingly up to 42; the blood-pressure descended as low as 78. These signs of medullary edema progressed so rapidly that an operation was not attempted and the patient died 3 hours after admission—the heart continuing to beat fully 4 minutes after the respiration had ceased.

The coroner's physician, Doctor T. D. Lehane, after hearing the history and taking one glance at the body in the hospital morgue, made the diagnosis of "fracture of the skull with intracranial hemorrhage" and refused to perform an autopsy; there were no external evidences of head injury and yet this learned consultant could make his diagnosis without even touching the body—let alone performing an autopsy. Fortunately, however, the parents of the boy insisted that the cause of death should be accurately ascertained, as even the laity are becoming less and less impressed by the ability of doctors, as well as of coroner's physicians, to make "snap" diagnoses. At the urgent request, therefore, of police headquarters, Doctor Lehane did perform an autopsy the following morning at the city morgue to which the body had been taken.

Autopsy.—Left temporal muscle very hemorrhagic and there was found a small linear fracture of the underlying left squamous bone about 4 cm. in length (Fig. 91). The dura was not torn and there was no subdural nor cerebral hemorrhage, but the brain itself was very much swollen and edematous—the typical "wet," "water-logged" brain. Ventricles negative. A little cerebrospinal fluid was slightly blood-tinged but there was not sufficient blood to cause an increased intracranial pressure. The medulla itself was swollen and edematous. No other injuries to the brain or skull were demonstrable at autopsy.

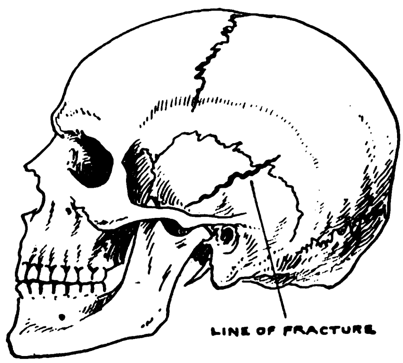


FIG. 91.—Small linear fracture of the squamous portion of left temporal bone in a patient who died three and one-half hours after the cranial injury from an acute medullary edema, due to an extreme intracranial pressure of cerebral edema alone.

Remarks.—This case is most instructive in that it illustrates again that an acute medullary compression and its resulting edema can occur in brain injuries, even in young adults, and of sufficient degree to cause death even in the absence of an intracranial hemorrhage. This extreme condition is unusual, I believe, in young adults without there being associated with it an intracranial hemorrhage; it more frequently occurs in patients beyond middle age and especially in those patients of lessened resistance due to chronic alcoholism and its allied conditions of arteriosclerosis and nephritis. If the condition of this patient could have been retarded so that the onset of medullary edema would have been delayed for a period of 2 hours or more, it is possible that a subtemporal decompression, and if necessary, a bilateral decompression would have sufficed to obtain a recovery of life—especially since the patient was a youthful adult and apparently in good health at the time of the injury.

I mentioned the details of the coroner's examination chiefly for the purpose of illustrating the difficulties, at times, of securing an official permission for performing a post-mortem examination upon these patients. The reluctance of many of the coroner's physicians to perform an autopsy even in doubtful cases where the diagnosis cannot be accurately determined without such an examination, and their apparent willingness to accept the history as the most important factor of the cause of death in many of these patients is most discouraging to the staff of a hospital who are naturally more interested beyond the point of knowing that the patient died. The too common diagnosis of "fracture of the skull" is by no means a sufficient cause of death in itself, and it is this very diagnosis "fracture of the skull" that has retarded the development of the rational treatment of brain injuries possibly more than any other factor. There have been notable exceptions among the coroner's staff of physicians, particularly that of Doctor O. H. Schultze, Doctor John McAllister and of Doctor Benjamin Schwartz, who are really interested in reaching the "real" diagnosis of the pathological condition in these patients and whose observations and suggestions have been most helpful to the attending physician and surgeon.

Case 66.—Acute severe brain injury associated with high intracranial pressure due to cerebral edema. No operation. Medullary edema; death. Autopsy.

No. 299.—John. Fifty-seven years. White. Married. Laborer. Ireland. Admitted June 28, 1915, Polyclinic Hospital.

Died July 1, 1915—61 hours after admission and 62 hours after injury.

Family history negative.

Personal history negative, except for chronic alcoholism.

Present Illness.—Patient is said to have fallen headforemost upon the street curbing while intoxicated; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (55 minutes after injury).—Temperature, 97.4°; pulse, 90; respiration, 28; blood-pressure, 110. Profound unconsciousness and in a severe degree of shock. Strong odor of whiskey upon breath of patient. Profuse bleeding from nose and both ears, but an ecchymosis only over the left mastoid area. Both orbits ecchymosed with bilateral subconjunctival hemorrhages. Pupils—both dilated, left larger than right. Reflexes—patellar increased but equal; no ankle clonus but double Babinski; abdominal reflexes absent. Fundi negative.

Treatment.—Expectant palliative; vigorous anti-shock measures instituted. The general condition of the patient improved so that 7 hours after admission the pulse had descended to 74, respirations to 22, while the blood-pressure had ascended to 126. This improved condition did not remain longer than one hour, when the temperature quickly arose to 104°, pulse to 122, respirations to 30, while the blood-pressure now descended to 108. The following examination was made at this time:

Examination (8 hours after admission).—Temperature, 104.4°; pulse, 126; respiration, 32; blood-pressure, 104. Profound unconsciousness and in apparent medullary collapse. Bleeding from both ears has ceased; otoscopic examination reveals a laceration of posterior portion of left tympanic membrane; although there is blood in the right auditory canal no laceration of the right tympanic membrane is visible; left mastoid ecchymosis present but right mastoid area is negative. Pupils widely dilated and do not react to light. Reflexes all depressed but apparently equal; double Babinski still persists. Fundi—retinal veins enlarged; nasal margins of both optic disks blurred by edema. Lumbar puncture—clear cerebrospinal fluid with an occasional streak of blood under high intracranial pressure (approximately 19 mm.).

Treatment.—The condition of medullary edema had occurred so rapidly following the lessening of the shock that at no time were the signs of acute medullary compression to be ascertained clinically, and therefore no operative relief of the intracranial pressure could be offered in the hope of retarding the onset of an acute medullary edema. The patient was treated expectantly in the forlorn hope that the signs of medullary edema would disappear, but the general condition of the patient progressively became worse—temperature reaching 106.8°, pulse 146, respiration 42, while the blood-pressure descended to below 86 within 36 hours after admission, and the patient finally died 61 hours after admission.

Autopsy (Doctor John McAllister).—Small linear fracture of 5 cm. long in squamous portion of left temporal bone extending obliquely downward into mastoid and petrous portions (Fig. 92). No extra- nor subdural hemorrhage. Over anterior portion of left cerebral hemisphere was a very thin film of subarachnoid hemorrhage—in all not more than a teaspoonful. Brain itself very swollen and "water-logged" and no punctate hemorrhages or lacerations in it; medulla also boggy and "wet." Ventricles negative. Examination of heart, lungs, liver and kidneys was practically negative.

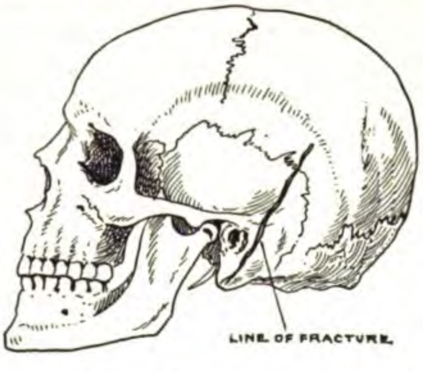


FIG. 92.—Small linear fracture of left vault and base in a patient precipitated early into the condition of acute medullary edema as the result of an extreme intracranial pressure due to cerebral edema.

Remarks.—This case is most interesting in that the coroner had great difficulty in ascertaining the cause of death—so much so that during the autopsy he frequently complained that a sufficient cause of death did not seem to be present since with the exception of a mild interstitial nephritis, hepatic fibrosis in addition to a slight valvular cardiac lesion, the findings at autopsy were practically negative—unless the "wet" edematous swollen condition of the brain was the immediate cause of death by means of an acute medullary edema. This latter belief was undoubtedly the explanation for the death of this patient having a severe head injury and upon admission to the hospital being in the condition of shock (temperature subnormal, pulse 90 and a blood-pressure of only 110); as the shock lessened, the temperature ascended to 101°, the pulse descended to 74, while the blood-pressure increased to 126—that is, the signs of an increased intracranial pressure overshadowing those signs of shock. However, the resistance of the patient to any increase of the intracranial pressure was so impaired that these mild signs of medullary compression resulting from the increased intracranial pressure quickly changed into the signs of acute medullary edema—the temperature rising rapidly to 104.4°, pulse to 126, while the blood-pressure descended quickly to 100 and lower. This patient did not die from shock, as a marked increased intracranial pressure is, in my opinion, impossible in the presence of extreme shock, and this patient did have a high intracranial pressure as accurately demonstrated by the lumbar puncture and, to a lesser degree, by the ophthalmoscopic examination of the fundi—a less accurate means of determining the intracranial pressure.

The fracture of the skull opening into the left auditory canal offered a means of lessening the increased intracranial pressure by a sort of natural decompression; in many patients, this natural drainage is sufficient to lower the intracranial pressure and thus an operation is avoided, but in the majority of patients the outlet for the escape of blood and cerebrospinal fluid through the fracture in the ear becomes blocked early, and thus a further lessening of the pressure is not possible. The danger of infection also through this channel must always be considered, and yet if no meddlesome cleaning of the external auditory canal is attempted and merely a sterile gauze pad applied loosely to the lobe of the ear, the risk of infection is really slight.

B. *Fracture not present.*

a. High intracranial pressure due to large hemorrhage.

Case 67.—Acute severe brain injury associated with signs of high intracranial pressure due to large hemorrhage. No operation. Medullary edema; death. Autopsy.

No. 153.—Charles. Fifty-eight years. White. Married. Steamfitter. Germany.

Admitted July 3, 1914, Polyclinic Hospital. Referred by Doctor R. E. Brennan.

Died July 5, 1914—30 hours after admission and 30½ hours after injury.

Family history negative.

Personal history negative.

Present Illness.—While riding upon an automobile truck, patient was thrown to the street as the result of a collision; momentary loss of consciousness; brought to the hospital in the automobile.

Examination upon admission (25 minutes after injury).—Temperature, 99.4°; pulse, 68; respiration, 20; blood-pressure, 130. Perfectly conscious, although the patient does not recall the accident; no alcoholism. Multiple lacerations and contusions of entire body. Fracture of right lower jaw. No bleeding from nose, mouth or ears; otoscopic examination negative. Pupils equal and react normally. Reflexes negative. Fundi—retinal veins enlarged; nasal margins of both optic disks blurred. Lumbar puncture—very bloody cerebrospinal fluid under increased pressure (approximately 16 mm.); upon allowing the cerebrospinal fluid to stand, almost 50 per cent. of it was blood.

Treatment.—Expectant palliative. The intracranial pressure did not seem to be sufficiently high enough to warrant an immediate operation of decompression and drainage—it was hoped that the patient could "take care of" this increased pressure and blood by the natural means of absorption. Patient was frequently examined, and the general condition improved, although the pulse remained between 64 and 68 and the ophthalmoscopic examinations revealed an increasing intracranial pressure so that both optic disks were entirely obscured by edema, but no measurable swelling of the disks—that is, a papilledema of mild degree; at this examination 15 hours after admission, a lumbar puncture was performed, allowing bloody cerebrospinal fluid to escape under high pressure (approximately 18 mm.). As there were no localizing signs, and as the reflexes still remained negative without a Babinski and as the general condition of the patient seemed so good in that he remained *perfectly conscious* and did not complain of severe headache, it was considered advisable to watch him carefully and if the signs of high intracranial pressure became more marked and his general condition became worse, then the operation of subtemporal decompression and drainage would be advised. (We now know that this attitude is not conservatism but rather one of ignorance and that it would have been and is always a much safer procedure to perform the operation of subtemporal decompression and drainage early in the presence of the definite signs of increased intracranial pressure as recorded by the ophthalmoscope and the spinal mercurial manometer, and that it is not necessary to have profound unconsciousness, changes of reflexes, so-called "localizing" signs, a very slow pulse with Cheyne-Stokes respiration and a high blood-pressure in order that the operation of cranial decompression be advised; these latter signs frequently do not occur and very frequently when they do occur, they appear so late in the progress of medullary compression that a medullary edema is very easily precipitated.)

Patient remained in practically the same excellent condition of consciousness with no definite complaints until the morning of July 5, at one o'clock, when he suddenly awakened from a heavy sleep, became very noisy and irrational, got out of bed, stood up, and upon being put into bed and restrained, he became comatose within 10 minutes, his pulse ascended to 148, while the blood-pressure dropped to below 90; ophthalmoscopic examination revealed dilated retinal veins with both optic disks blurred to the measurable swelling of 2 diopters—that is, the early stage of "choked disks." Respiration stopped 10 minutes later, and although artificial respiration was performed and the heart continued to beat for 6 minutes, yet the patient died—a death of typical medullary edema.

Autopsy.—Small laceration of scalp over posterior occipital area. No fracture of the skull could be found. No extradural hemorrhage. Large subdural hemorrhage of dark syrupy character and about one-fourth inch in thickness over both cerebral hemispheres. No cortical hemorrhage nor laceration. Much free blood and cerebrospinal fluid subtentorially about the medulla. Ventricles negative.

Remarks.—This patient naturally should have been operated upon—a subtemporal decompression and drainage used, and if necessary, a bilateral decompression, within 15 hours after admission, when the signs of an increased intracranial pressure appeared both in the fundi and at lumbar puncture, rather than waiting for unconsciousness and localizing signs or a very slow pulse, Cheyne-Stokes respiration and a high blood-pressure. To allow patients of this character to attempt "to take care of" intracranial pressure of this severity is a most risky procedure—a far greater danger to the patient than the operation of decompression performed under modern conditions of asepsis and technic.

It is indeed surprising that the patient, in view of the autopsy findings, did not have more signs of cortical irritation and even of cortical compression; not only were the reflexes not increased and no Babinski present, but the pupils were of normal size and of normal reaction to light, and the only sign of cortical irritation was possibly the extreme restlessness of the patient—although this was not more than is ordinarily observed in many patients.

Three hours before death, an ophthalmoscopic examination had been made and the report of the house surgeon was that a papilledema of one diopter was present; undoubtedly the operation should have been performed at this late date, but it was still believed that the patient would recover without an operation—"the general condition was so good, the pulse only 66, and not even a Babinski."

The absence of a fracture of the skull merely emphasizes the relative unimportance of the fracture in patients having brain injuries, unless it is remembered that the fracture frequently offers a means of escape for blood and cerebrospinal fluid, and thus a natural method of lessening an increased intracranial pressure is afforded so that an operation may be avoided; the most severe cases, as this patient illustrates, are very frequently not even associated with a fracture of the skull.

b. High intracranial pressure due to cerebral edema alone, and no intracranial hemorrhage present.

CASE 68.—Acute severe brain injury associated with signs of high intracranial pressure due to cerebral edema alone. No operation. Medullary edema; death. Autopsy.

No. 965.—Unknown woman. About fifty years. White.

Admitted March 14, 1918, Polyclinic Hospital.

Died March 16, 1918—42 hours after admission.

Family history not known.

Personal history not known.

Present Illness.—While crossing the street in an intoxicated condition, patient was knocked down by an automobile; immediate loss of consciousness; brought to the hospital in the automobile.

Examination upon admission (20 minutes after injury).—Temperature, 97.8°; pulse, 50; respiration, 24; blood-pressure, 112. Profound unconsciousness. Well-nourished and rather obese plethoric woman of about 50 years of age. Laceration of scalp over left frontal area of 4 inches in length; careful probing revealed no underlying fracture or depression. Bleeding profusely from left ear; extensive left mastoid ecchymosis. Pupils equally dilated and do not react to light. Reflexes—patellar exaggerated but apparently equal; no ankle clonus nor Babinski; abdominal reflexes absent. Fundi—retinal veins enlarged and a general haziness of entire retinæ not limited to optic disks. Lumbar puncture—clear cerebrospinal fluid under normal pressure (8 mm.).

Treatment.—Expectant palliative; vigorous anti-shock measures instituted. However, within 2 hours after admission the temperature had ascended to 101°, while the pulse had risen rapidly to 126, respiration to 36, while the blood-pressure had decreased to 104. The general condition of the patient rapidly became worse so that 24 hours after admission the temperature was 105°, pulse 142, respiration 40 and the blood-pressure 92. Patient became markedly cyanotic and the signs of pulmonary edema developed, so that rattling moist râles occurred throughout both lungs; in spite of most active stimulative treatment, the condition of the patient became worse and she finally died 42 hours after admission from a typical medullary edema.

Autopsy.—Multiple ecchymoses of the scalp, especially in the left mastoid area; no fracture of the skull ascertained. The cerebral hemispheres were very "wet" and "water-logged," but no gross subdural or intracerebral hemorrhages found. Much clear cerebrospinal fluid subtentorially about the medulla. Anterior surface of right frontal lobe slightly contused with an occasional punctate hemorrhage in several places. Ventricles negative.

Remarks.—Upon admission, the subnormal temperature and low blood-pressure indicated a high degree of shock; and yet, if the ophthalmoscopic examination (which was practically negative, or at least did not show the signs of a high increased intracranial pressure) and the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer (which indicated an intracranial pressure of only 8 mm.) had not been practically negative, it might have been believed that the general intracranial pressure was high and that it was the cause of the lowered pulse-rate of 50 in the presence of severe shock. I do not believe this was the case, but rather it was the condition of an acute direct medullary compression itself in a middle-aged woman having chronic alcoholism and that, as the subsequent history of the case shows, this acute medullary compression changed very rapidly into the condition of acute medullary edema and therefore the early death of the patient. It would be most rare for a general intracranial pressure to produce an acute medullary compression sufficient to lower the pulse-rate to 50, and yet there not be these signs of a high intracranial pressure as revealed by the ophthalmoscope, lumbar puncture and to a less accurate extent the blood-pressure itself. Besides it would be most difficult, and I doubt if it can happen, for extensive bleeding to occur intracranially—at least to an amount sufficient to increase the general intracranial pressure so that an acute medullary compression would occur—in the presence of shock to the extent of causing a subnormal temperature of 97.8° and a blood-pressure of only 112.

A possible explanation that the lowered pulse-rate of 50 in this patient did indicate high intracranial pressure with a medullary compression, complicated clinically by the signs of severe shock, and that the lumbar puncture did not indicate an increased intracranial pressure because the cerebrospinal fluid was blocked at the foramen magnum by the medullary compression itself, is, in my opinion, not warranted, chiefly because the ophthalmoscopic examination of both fundi was practically negative.

I do not believe that this patient could have recovered under any circumstances—operation or no operation—because the progress of the con-

dition with the onset of an acute medullary edema was s
treatment could be of any benefit.

ACUTE SEVERE BRAIN INJURIES ASSOCIATED WITH A HIG PRESSURE DUE TO HEMORRHAGE AND CEREBRAL EDEMA DECOMPRESSION. DEATH; AUTOPSY.

In this group of patients, with and without a fracture o
operation of subtemporal decompression was advised as th
means of lowering the high intracranial pressure of hemorrhag
edema and thus affording the patient a chance of recovery; wi
operation, almost all of these patients die or, if an exceptional p
recover life under the expectant palliative treatment, then they
never regain their former good health and normality—both phy
mentally, and also emotionally. The risk of the operation is
pared with the gravity of the condition of these patients. When
of severe initial shock disappear, if it is determined that the in
pressure is increasing, as is best indicated by the ophthalmoscopic
manometric tests as well as by the gradual descent of the pulse- an
tion-rates and the ascent of the blood-pressure, then the conditio
patient should be most carefully and repeatedly observed in the h
the expectant palliative treatment alone will suffice to prevent a hig
cranial pressure; but if it is ascertained that the intracranial p
continues to rise beyond the limits adequately treated by the expectant
tive method—such as an increased intracranial pressure sufficient to p
a papilledema or even an edematous blurring of the nasal halves an
poral margins of the optic disks and the pressure of the cerebrospin
at lumbar puncture registers a height of 16 mm. and over, and esp
if the pulse- and respiration-rates are 60 and 16 and lower, respe
then there should be no delay in advising an immediate subtemporal
pression and drainage, and if at this operation the increased intra
pressure is so high that the bulging cortex does not pulsate or only
so and comparatively little hemorrhage and cerebrospinal fluid are
owing to the "water-logged" swollen condition of the brain, then an
diate bilateral decompression should be advised and performed.

The high mortality in these patients is due not only to the extrem
cranial condition but also, and chiefly, to a delayed lowering of the in
intracranial pressure so that the patient is in a most hazardous co
of severe medullary compression before the operation is attemp
allow these patients to develop definite signs of medullary com
with very low pulse- and respiration-rates of even 50 and 14 and lo
spectively, and of the Cheyne-Stokes character of rhythmical irregul
when the onset of an acute medullary edema may occur at any mom
therefore the end of the patient within a few hours—in the hope
belief that the patient can still "take care of" the condition, this
is a most mistaken one and cannot be too strongly condemned; n
there are patients, and we have had them in this series of cases,
recover life from this extreme and dangerous condition of severe me
ssion with the expectant palliative treatment alone, but
how that the percentage of recovery of life alone is less tha

nor became worse in the next 2 days, but then the condition changed markedly as shown by the following examination:

Examination (44 hours after admission).—Temperature, 102° ; pulse, 68; respiration 20; blood-pressure, 130. Patient still semiconscious—easily aroused but will not speak. Large occipital hematoma. Pupils equal and react normally. Reflexes: patellar—left greater than right; no ankle clonus nor Babinski; abdominal reflexes both depressed but equal. Fundi—retinal veins dilated; nasal halves of both optic disks blurred. Lumbar puncture—bloody cerebrospinal fluid under high pressure (approximately 18 mm.).

Treatment.—Right subtemporal decompression and drainage advised immediately to lessen the increasing intracranial pressure.

Operation (48 hours after admission).—Right subtemporal decompression: usual vertical incision, bone removed and no complications. Dura very tense and extending down over the upper third of the operative field was an extradural hemorrhage, one-quarter inch in thickness; this clot extended upward beneath the parietal bone. Upon incising the dura, bloody cerebrospinal fluid spurted and upon enlarging the dural opening a very "wet" edematous cortex tended to protrude under pressure, but did not rupture; arachnoid "sweating" was very profuse. On account of the high cerebral tension, a ventricular puncture was performed, but clear cerebrospinal fluid under normal pressure escaped. In spite of the loss of a large quantity of cerebrospinal fluid and free blood, yet the cerebral tension remained quite high and only slight cerebral pulsation was visible. Usual closure with 2 drains of rubber tissue inserted. Duration, 50 minutes.

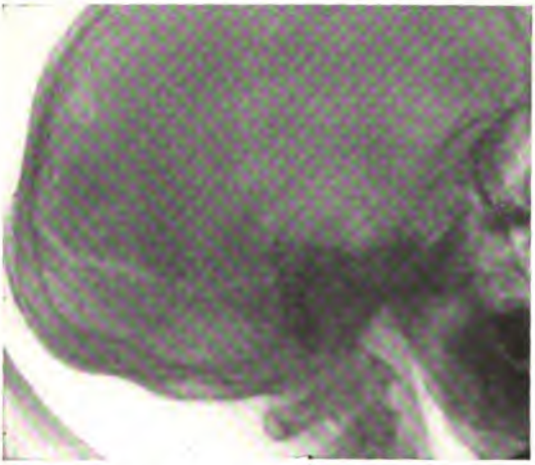


Fig. 93.—Wide linear fracture of the "bursting" type of the right half of the occipital bone in a patient having a high intracranial pressure, due to extradural and subdural hemorrhage. Right subtemporal decompression and drainage. Death from medullary edema.

Post-operative Notes.—Patient did not become conscious after operation; and he became weaker and weaker. Ten hours after operation the temperature was 102.4°, pulse 130, respiration 44, and the blood-pressure 115—that is, medullary edema had occurred, and naturally the death of the patient—24 hours after operation. X-ray (obtained post-mortem by Doctor A. J. Quimby)—"reveals a wide linear fracture extending from the occipital protuberance into the right mastoid area; also right decompression is shown faintly" (Fig. 93).

Autopsy.—Large hematoma 2 inches in diameter beneath the periosteum over right occipital bone; several small hematomata in right frontal region.

Fracture extended around the head horizontally from the occipital protuberance into right mastoid area and also to the left to a point one-half inch above the left external auditory meatus; this fracture was of the typical "bursting" type, being of special interest in that the line of fracture of the outer table was always very prominent and wider than the internal line of fracture which was even absent in several places (Fig. 94). Several small lines of fracture radiated from this main horizontal fracture. Left frontal lobe and upper part of left temporo-sphenoidal lobe lacerated over an area of 2 inches in diameter. Subdural hemorrhage over left frontal lobe. No cortical hemorrhage nor laceration over right side of brain. Ventricles normal; no basal hemorrhage. The entire brain, however, was very much swollen with edema, "water-logged," and it was this edema with its resulting pressure which caused the death of the patient through medullary compression and the subsequent medullary edema.

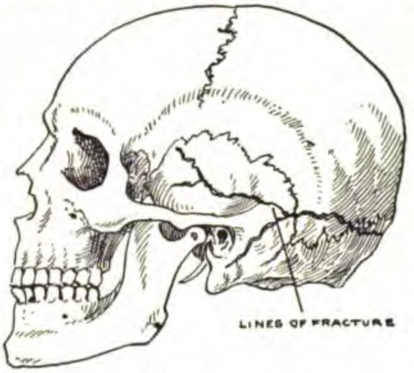


FIG. 94.—The left portion of the horizontal linear fracture of the posterior half of the vault. The line of fracture was characteristic of the "bursting" type of fracture in that the outer table was more widely separated than the inner table and frequently the inner table was not even fractured at all although the overlying table was definitely separated.

Remarks.—An earlier relief of the intracranial pressure might have afforded this patient a chance of recovery; the alcoholism so masked the true intracranial condition that the symptoms and signs of intracranial pressure were not observed as having developed until 44 hours after admission; by this time the signs of medullary edema were already present—that is, the pulse was rising rapidly from 50 to 68, the respirations from 16 to 20, and the blood-pressure was falling from 140 to 130; within 10 hours after operation, the condition of medullary edema had so far advanced that the pulse had ascended to 130, respiration to 44 and the blood-pressure had fallen to 115. That is, this patient, instead of being operated upon earlier in the stage of medullary compression in its milder form, had been allowed to advance so that the pulse had reached its lowest level before the signs of high intracranial pressure had been determined, and when they had been ascertained, then an operation had been advised when the signs of medullary edema were present; these patients almost invariably die once they have developed the definite signs of medullary edema, and so it happened in this patient—operation or no operation. The mistake made in treating this patient was due to the observation that there was the odor of alcohol upon his breath, and therefore he was considered an alcoholic with a bump on his head rather than a patient with a possible brain injury having the odor of alcohol upon his breath; alcoholic intoxication does tend to mask the symptoms and signs of an acute brain injury, but if the signs of intracranial pressure, as ascertained by the ophthalmoscopic examination of the fundi and in the measurement of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer are present, then these patients should be treated accordingly, whether alcoholism is a factor or not. This patient, however, did not show signs of an increased intracranial pressure by the ophthalmoscopic examination, and therefore no lumbar puncture was performed upon admission—the condition being considered one rather of intoxication than of brain injury; it is now realized that it is rare for the signs of increased intracranial pressure to be registered in the fundus of the eye within six hours after the injury, and therefore in all doubtful patients, the lumbar puncture is a most valuable and accurate means of determining the exact increase of the intracranial pressure, and it should always be performed in the absence of shock.

These "bursting" fractures of the vault are interesting in that frequently, as Doctor O. H. Schultze has mentioned, it is easily observable at autopsies that the middle portion of the "bursting" line of fracture or "crack" is much wider than either of the end portions, showing that the fracture is really the mechanical result of approximating the pole of contact and its opposite pole, so that it "bursts" or "cracks" in its thinnest or weakest meridian; it was true in this patient.

Case 70.—Acute severe brain injury associated with high intracranial pressure due to cerebral edema and subdural hemorrhage. Right subtemporal decompression and drainage. Death. Autopsy.

No. 852.—Arthur. Twenty-eight years. White. Married. Jockey. U. S.

Admitted May 4, 1917—2 hours after injury. Coney Island Hospital, Coney Island, New York. Referred by Doctor L. T. Smith.

Operation May 6, 1917—51 hours after admission. Right subtemporal decompression and drainage.

Died May 7, 1917—30 hours after operation.

Family history negative.

Personal history negative.

Present Illness.—While driving an automobile, the patient collided with another car and was thrown headlong to the street; immediate loss of consciousness; brought to the hospital in the ambulance; upon admission the pulse was 52 and there was profuse bleeding from both ears and profound unconsciousness; an immediate operation was advised and a small trephine opening was made 5 hours after admission over the right parietal area, but the dura was not opened—merely a hypodermic needle inserted and a small amount of bloody cerebrospinal fluid removed. At the end of the operation the pulse was 64, and 3 hours later it was 70, and within 10 hours after admission the pulse had rapidly risen to 128, where it remained during the next 6 hours, and then descended and varied from 112–124 until the following examination:

Examination (48 hours after admission; in consultation with Doctor Smith).—Temperature, 100.8°; pulse, 112; respiration, 28; blood-pressure, 126. Profound unconsciousness. Right eye extensively ecchymosed and much clotted blood in both auditory canals, and there is still a slight bloody discharge from right ear; boggy bilateral mastoid ecchymosis. Pupils—right larger than left and reacts to light sluggishly. Reflexes: patellar—left greater than right; double ankle clonus and double Babinski; abdominal reflexes absent. Fundi—retinal veins dilated, blurred and tortuous;

both nasal and temporal halves of optic disks obscured by edema but no measurable swelling to the extent of one diopter. Lumbar puncture—bloody cerebrospinal fluid under high intracranial pressure (approximately 17 mm.).

Treatment.—It was a most difficult question to decide whether a subtemporal decompression should be advised at this late period or whether the condition should be considered hopeless and no operation considered; yet at the time, I considered it a borderline case from the operative standpoint and I felt that an operation might give the patient a definite chance to recover (and in this opinion I was mistaken). An immediate right subtemporal decompression was advised in the hope that a lessening of the intracranial pressure would permit the signs of medullary edema to subside and thus the recovery of the patient be obtained.

Operation (51 hours after admission).—Right subtemporal decompression (no anesthesia being necessary): usual vertical incision, bone removed and no complications; lower branch of right middle meningeal artery bled freely until successfully blocked with bone wax. Dura very tense and bluish, and upon incising it, dark bloody cerebrospinal fluid spurted to a height of 2 inches; upon enlarging dural opening, much bloody cerebrospinal fluid welled out through opening, revealing a very "wet," congested, swollen brain which began to pulsate after the escape of much cerebrospinal fluid and free blood (it was observed at this time that the patient became less resistant and quieter, breathed less irregularly and not so stertorously, and also the pulse became better in quality but did not become less rapid). No cortical hemorrhage or laceration visible. Usual closure with 2 drains of rubber tissue inserted. Duration, 50 minutes.

Post-operative Notes.—Patient remained in practically the same condition as immediately before the operation, except that the general condition seemed improved; did not become conscious, however, and neither did the pulse nor respiration descend, although their character was improved; after 24 hours, however, the patient rapidly became worse—temperature ascended to 107°, pulse and respiration rose quickly to 150 and 38, respectively, and the patient died 30 hours after operation and 78 hours after injury from the typical condition of medullary edema and collapse.

Autopsy.—Fracture of base through both middle fossæ and both petrous portions of temporal bones; no fracture of vault ascertained (Fig. 95). No extradural hemorrhage and only a thin film of subdural hemorrhage mixed with cerebrospinal fluid. Anterior portion of right frontal lobe contused but not extensively. Brain itself very edematous and much cerebrospinal fluid subdurally. No cortical punctate hemorrhages or lacerations ascertained.

Remarks.—This patient would have had an excellent chance to recover if a subtemporal decompression had been performed immediately after the patient's admission to the hospital, when the pulse was 52—that is, presumably the stage of medullary compression as indicated by a very low pulse-rate. The operation which was performed at that time—a small trephine opening and the dura not opened, and particularly the use of a hypodermic needle thrust "blindly" through the dura—can hardly be considered an operation unless classed as meddlesome surgery with great danger to the patient; to remove an area of bone and not to open the dura widely does not permit a cranial decompression to occur, as the dura is inelastic in adults and the entire top of a patient's head—that is, the bony vault—could be rongeured away and yet, if the dura was not widely opened, no decompression or relief of the intradural pressure would be possible. The use of a hypodermic needle to ascertain the presence or not of subdural blood is not only distinctly dangerous but it can be of no aid in the treatment; it is not the presence of subdural blood which is necessary to be ascertained in the operative treatment or not but the presence or not of an increased intracranial pressure; beside the accurate tests preceding any cranial operation for estimating approximately the intracranial pressure—such as careful ophthalmoscopic examinations and the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer, and if an operation is considered advisable, then upon exposing the dura through a large bony opening it is very easily ascertained by palpation of the dura, whether the intradural pressure is high or not, and the appropriate procedure can then be performed. A small trephine opening, and particularly over the parietal area, is distinctly dangerous and unsurgical—if the dura is not opened then the operation itself is not warranted because no benefit can possibly result from such a procedure, and if the dura is opened and the intradural pressure is high, then there is the great danger of the underlying cerebral motor cortex being forced upward through the small dural and bony opening and thus producing a permanent damage to these nerve cells and resulting in paralysis, impairments of sensation, special senses and of speech itself; the danger also of post-traumatic sequelæ from such damage to the cerebral cortex must always be remembered. In a general way, in the presence of high intracranial pressure, a small bony opening of the vault (and the dura opened) is always much more dangerous and liable to complications than a large bony opening; many disasters in cranial surgery in the presence of high intracranial pressure have been, and are due to, a small and supposedly "safe" opening of the bony vault.

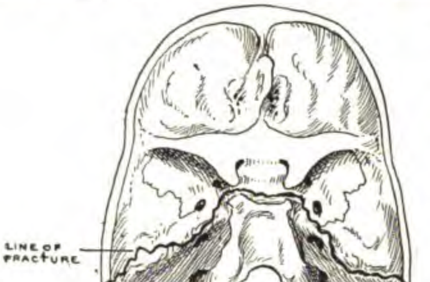


FIG. 95.—Extensive basilar fracture through both petrous bones and the sella turcica in a patient having a high intracranial pressure due to a subdural hemorrhage and cerebral edema. A right subtemporal decompression and drainage failed to prevent the progress of an advancing medullary edema and the death of the patient.

The operation of cranial decompression performed upon this patient occurred at too late a stage to prevent the usual progress of medullary edema—once it has begun; it is rare for a patient to recover once the pulse has descended to its lowest level of medullary compression and then when the pulse begins to rise rapidly and the blood-pressure to fall, indicating the onset of medullary edema, then it is exceedingly rare for the patient to recover—operation or no operation. In this patient, however, although the pulse had ascended within 10 hours from 52 to 128 (the respiration correspondingly, and the blood-pressure had descended), yet the pulse then descended slightly and remained between 112–124, until 38 hours later, when the consultation was held; it appeared that the patient was withstanding the effects of the medullary edema, at least temporarily, and it was hoped that the patient would have a greater chance of recovery if the intracranial pressure was lessened by a subtemporal decompression and drainage of the bloody cerebrospinal fluid. It was considered that the patient without an operation had no chance to recover from the medullary edema and that he might have a definite chance if the operation of cranial decompression was performed. My experience with similar patients does not confirm this belief, and I do not think that a cranial operation is justified in these patients who have passed through the stage of medullary compression and have advanced into the period of medullary edema where the pulse-rate is above 110 and the blood-pressure is below 120; it is indeed very difficult to refuse to operate upon these patients in the early stage of medullary edema—hoping against hope that the operation may give them a chance of recovery, but these patients recover so rarely that the operation is hardly justified and it is not creditable to modern surgery to advise cranial operations upon these patients in this condition of severe medullary edema.

The profuse discharge of blood through both ears undoubtedly lessened the increased intracranial pressure and delayed the onset of medullary edema; it is unfortunate that this drainage usually ceases within 12 hours after the injury, and although the danger of infection through the ear increases if the drainage is prolonged beyond 48 hours, yet the risk of such a complication is ordinarily a slight one if meddlesome treatment of blocking or irrigating the ear is not used.

CASE 71.—Acute severe brain injury with subdural hemorrhage associated with a marked degree of shock and medullary edema. Right subtemporal decompression and drainage. Death. Autopsy.

No. 051.—Daniel. Forty-one years. White. Married. Longshoreman. Ireland.

Admitted October 21, 1913, Polyclinic Hospital. Referred by John A. Bodine.

Operation October 22, 1913—25 hours after injury. Right subtemporal decompression and drainage.

Died October 23, 1913—18 hours after operation.

Family history negative.

Personal history negative.

Present Illness.—While intoxicated, patient was struck by a street car; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (40 minutes after injury).—Temperature, 98°; pulse, 120; respiration, 28; blood-pressure, 120. Rather obese and well-nourished; alcoholic. Profound unconsciousness; severe degree of shock. Extensive laceration over external occipital protuberance; gentle probing did not reveal an underlying fracture. Profuse bleeding from right ear; no cerebrospinal fluid observed. Pupils—right larger than left and does not react to light. Reflexes—patellar present and equal; no ankle clonus nor Babinski; abdominal reflexes cannot be obtained. Fundi negative.

Treatment.—Expectant palliative; vigorous anti-shock measures instituted. During the next 12 hours, patient's condition improved apparently so that he became semiconscious and could be easily aroused, but not sufficiently to answer questions; marked ecchymosis of right eye. Fourteen hours after admission, patient had a convulsive seizure of left side of face and left arm, but left leg not involved; at this time, temperature was 101.4°, pulse 134, respiration 34, blood-pressure 120; reflexes and fundi same as at preceding examination. Sixteen hours after admission, temperature was 103.4°, pulse 140, respiration 40, blood-pressure 110, and patient had another convulsion limited to left side of face, left arm, and left leg, and lasting 3 minutes. Fundi—retinal veins dilated; nasal margins of both optic disks blurred by edema. Nineteen hours after admission, another convulsion occurred limited to left side of body, and then similar convulsions occurred every 15 minutes until 22 hours after admission, when they were finally checked by the use of chloroform, morphia, bromides and chloral per rectum; at this time, patient was in extremis so that atropine, cupping, intravenous saline injections and extensive stimulation were used to prevent the appearance of a pulmonary edema and to raise the blood-pressure which had descended to a 104. Through a mistaken conception that the patient might be given a chance to recover if a subtemporal decompression was performed, even in this advanced stage of medullary edema and although there were no signs of pressure to be observed either by an ophthalmoscopic examination of the fundi or by lumbar puncture, yet the operation was advised. (We now know that any cranial operation performed during the period of initial and severe shock following head injuries or during the period of medullary edema, and especially during the more advanced stages of medullary edema, that any cranial operation during these two periods tends to take away whatever chances the patient has to recover, and although without an operation a patient in medullary edema has little or no chance of recovery, yet if an operation is performed during this period the death of the patient is merely hastened.)

Operation (24 hours after admission).—Right subtemporal decompression: usual vertical incision, removal of bone and no complications. Dura moderately tense and bluish, and upon incising it blood-tinged cerebrospinal fluid welled out through the dural opening, revealing a very "wet," edematous cortex, but not under sufficiently high tension to cause it to protrude; many subdural clots. Ventricular puncture removed almost three ounces of blood-tinged cerebrospinal fluid, but not under high pressure. Much cerebrospinal fluid escaped and the cortex pulsated rapidly and feebly at end of operation—the patient being in poor condition. Usual closure with 2 drains of rubber tissue inserted. Duration, 45 minutes.

Post-operative Notes.—Patient did not react after the operation but became gradually worse, so that 12 hours after operation the temperature was 106°, pulse 160, respiration 34, blood-pressure 90; patient died 18 hours after operation from the condition of typical medullary edema and collapse.

Autopsy.—Line of fracture radiated from external occipital protuberance downward in median line across the foramen magnum and upward through basilar process anteriorly to the posterior clinoid process of the sella turcica (Fig. 96). Ecchymosis of both orbits and bilateral subconjunctival hemorrhages; between the orbital periosteum and bone there were several small hemorrhages in both orbits—more in right than in left, and this accounted for the bilateral subconjunctival hemorrhages and orbital ecchymoses; there was, however, no fracture of the orbital bones themselves. Clotted blood in the right external auditory canal with a laceration of the right tympanic membrane and clotted blood in the right middle ear and yet no fracture of the petrous bone, although there was a small clot above the tegmen tympani. No extradural hemorrhage; bloody subdural cerebrospinal fluid with extensive laceration and supracortical hemorrhage of both frontal lobes, particularly the left (Fig. 97). Much hemorrhage and subdural clots subtentorially about the cerebellum, and especially over the posterior surface of the medulla.

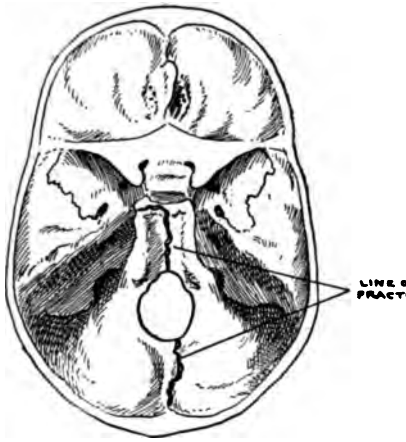


FIG. 96.—Wide linear fractures of the occipital bone and extending into the foramen magnum—the most dangerous type of cranial fracture on account of the frequent subtentorial complication of direct medullary compression and the resulting edema—as occurred in this patient.

Remarks.—This case clearly illustrates the futility of operating upon patients who have progressed into the stage of medullary edema and these patients should never be operated upon in the mistaken belief that the operation may give the patient a chance to recover. Operations performed in this stage of medullary edema and also in the stage of severe shock immediately following so many cranial injuries tend to discredit cranial surgery as the mortality is very high, and in the condition of extreme shock a cranial operation tends to lessen the patient's chance of overcoming the shock and thus in reality takes away to a large extent the patient's chance of surviving the shock.

This case also illustrates the great danger of fractures of the skull occurring beneath the tentorium; any cranial injury sufficient to cause a fracture beneath the tentorium is most liable to cause a definite brain injury subtentorially, either directly or by hemorrhage and edema, and thus increasing the subtentorial pressure and consequently a direct medullary compression with the great danger of an early medullary edema and collapse; the medullary edema may occur so rapidly after the disappearance of the signs of shock that the condition of shock may merge directly into that of medullary edema without apparently progressing through the condition of medullary compression—at least clinically; and so it happened in this patient—upon admission the condition was one of shock with a temperature of 98° and a pulse of 120; during the next 12 hours the patient recovered somewhat from the condition of shock only to pass into the stage of medullary edema with a temperature of 101° and a pulse of 134, and then the usual history of medullary edema—the temperature rising rapidly, the pulse- and respiration-rates also ascending, while the blood-pressure descends. At no time during the examinations of this patient could the signs of high intracranial pressure be ascertained and thus from this standpoint again no operation was indicated—no matter how badly the skull might be fractured—if there was no increased intracranial pressure, the operation could surely not be a decompression and therefore there could be no logical reason why an operation should be performed. Undoubtedly, the patient could not bleed intracranially to any extent on account of the lowered blood-pressure of shock, and then whatever subtentorial bleeding occurred exerted such high and rapidly-forming direct pressure upon the medulla that medullary edema was produced unusually early—as so frequently happens in cases of subtentorial fractures of the skull or rather acute subtentorial lesions producing a rapid increase of the subtentorial pressure. In a small percentage of patients having increased subtentorial pressure associated with or without an occipital fracture in cases of cranial injury, the suboccipital decompression may be indicated when the signs of medullary compression are present, due more to the local condition of increased subtentorial pressure than to a high increase of the general intracranial pressure—in which latter patients the subtemporal decompression is always to be preferred. It is rare, however, in acute brain injuries for a simple medullary compression due to direct subtentorial pressure to occur and to remain uncomplicated by a medullary edema for a sufficiently long time to permit a suboccipital decompression to be performed; not only is the suboccipital decompression a much more formidable operation than the subtemporal decompression, but in these acute subtentorial lesions, medullary edema occurs so quickly that the patient is really moribund before he can be prepared for an operation—let alone the performance of the operation itself; in chronic lesions subtentorially, the increase of subtentorial pressure is so gradual, as in cerebellar and in cerebello-pontine angle tumors, that the signs of

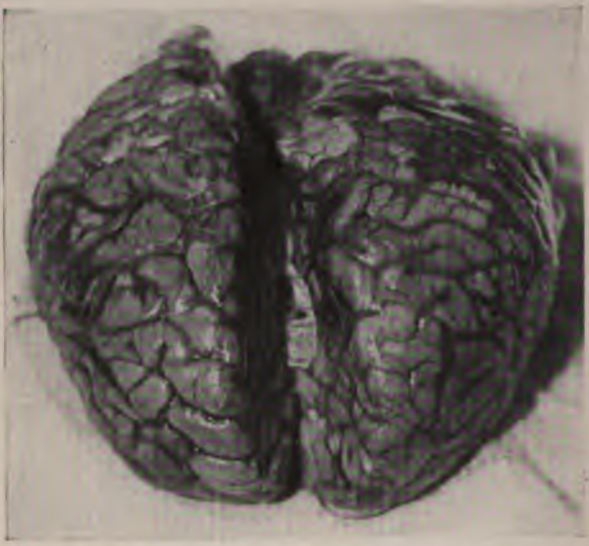


FIG. 97.—Extensive supracortical and subarachnoid hemorrhages of both cerebral hemispheres, left possibly more than right. Both frontal lobes lacerated, especially upon their inferior surfaces.

thus designated as the condition of "choked disks." A lumbar puncture would merely reveal the presence or not of blood in the spinal cerebrospinal fluid and that information is of no importance in the treatment—the presence or not of a high increase of the intracranial pressure being the all-important factor in the method of treatment, whether the expectant palliative or the operative treatment being advisable.

The ventricle puncture revealing the presence of a ventricular hemorrhage made the prognosis of this patient a most serious one, as this complication usually causes a fatal termination early.

Case 72.—Acute severe brain injury having marked signs of high intracranial pressure due to subdural hemorrhage and cerebral edema. Left subtemporal decompression and drainage. Death. Autopsy.

No. 670.—James. Twenty years. White. Single. Chauffeur. U. S.

Admitted August 29, 1916, Polyclinic Hospital.

Operation August 29, 1916—10½ hours after injury. Left subtemporal decompression and drainage.

Died August 30, 1916—24 hours after operation.

Family history negative.

Personal history negative.

Present Illness.—While driving his automobile, patient collided with another car and was hurled headlong to the street; immediate loss of consciousness; brought to the hospital in the automobile.

Examination upon admission (20 minutes after injury).—Temperature, 98°; pulse, 92; respiration, 28; blood-pressure, 118. Unconscious and in shock. Stellate laceration over left portion of occipital protuberance; careful probing revealed no underlying fracture. Small amount of blood trickled from left ear and nose. Pupils—enlarged and react sluggishly to light. Reflexes—negative except that they were all depressed. Owing to the shock no further examination was made at this time.

Treatment.—Expectant palliative; vigorous anti-shock measures instituted.

Examination (8 hours after admission).—Temperature, 104°; pulse, 74; respiration, 18; blood-pressure, 138. Patient still unconscious but has become very restless, requiring restraint. Still bleeding slightly from left ear. Pupils—right larger than left and does not react to light. Reflexes: patellar—right greater than left; no ankle clonus but suggestive right Babinski; abdominal reflexes—right absent and the left are very sluggish. Fundi—retinal veins dilated; both optic disks entirely obscured by edema with a measurable swelling of 1–2 diopters—that is, the condition of papilledema but not to the degree of "choked disks." Lumbar puncture—bloody cerebrospinal fluid under high pressure (approximately 18 mm.).

Treatment.—On account of the early rise of the intracranial pressure so that the condition of shock was entirely overshadowed by the signs of intracranial pressure, a subtemporal decompression was advised for fear that an early medullary edema would occur; the presence of a suggestive right Babinski with increased right deep reflexes and diminished right superficial reflexes made a left subtemporal decompression advisable, although the dilated right pupil would tend to make a right subtemporal decompression to be preferred—as it always is in right-handed patients and where the localizing signs are not definite or are even confusing.

Operation (10 hours after admission).—Left subtemporal decompression: usual vertical incision, bone removed and no complications. Dura very tense and bluish, and upon incising it, bloody cerebrospinal fluid welled out under very high pressure; upon enlarging the dural opening, there was exposed a supracortical layer of blood-clot of one-quarter inch in thickness. The cerebral tension was so high that the hernial protrusion caused a cortical rupture just beneath the Sylvian fissure; however, so much free blood and cerebrospinal fluid escaped that the cortex gradually receded and even pulsated slightly at the end of operation, so that a bilateral decompression was deemed unnecessary. Many punctate hemorrhages throughout the cortex. Usual closure with two drains of rubber tissue inserted. Duration, 55 minutes.

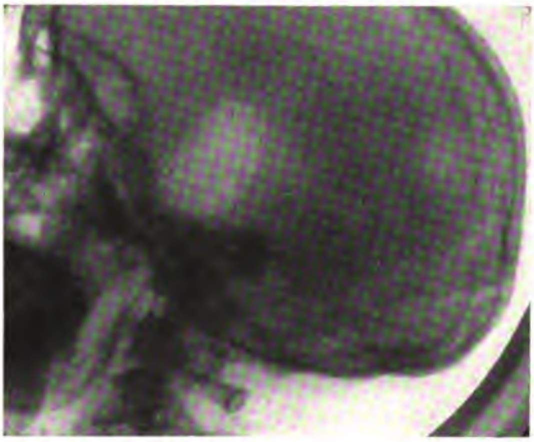


FIG. 98.—Wide linear fracture of left occipital bone, extending to the foramen magnum and through the petrous portion of the left temporal bone in a patient having a high intracranial pressure, due to subdural hemorrhage and cerebral edema. Left subtemporal decompression and drainage failed to prevent the onset of an acute medullary edema.

Post-operative Notes.—Patient seemed to improve during the first 10 hours following the operation in that the pulse did not descend below 70 and the blood-pressure remained above 104; patient became more conscious, but never sufficiently to answer questions; reflexes remained the same, however, and the fundi did not improve. Fifteen hours after operation, pulse began to ascend very rapidly to 114, temperature to 106°, respirations to 32 and the blood-pressure descended to 112. Twenty hours after operation, temperature was 107°, pulse 150, respirations 40 and blood-pressure 98—at this time, pulmonary edema became evident so that patient died three hours later—that is, 24 hours after operation—from the condition of typical medullary edema and collapse. An X-ray picture was taken postmortem, showing the occipital fracture, the left decompression opening and two silver clips (Fig. 98).

Autopsy.—Linear fracture extended from the external occipital protuberance downward on the left side to the foramen magnum and then upward and forward through the petrous portion of the left temporal bone into the middle fossa (Fig. 99). No extradural hemorrhage. Diffuse subdural hemorrhage over both hemispheres with extensive contusion and laceration of right frontal lobe—*"trauma au contra-coup."* No intracerebral or ventricular hemorrhage, but much free blood and cerebrospinal fluid in the subtentorial fossa about the medulla. Pituitary gland itself was definitely enlarged—the anterior lobe itself being the size of almost a small cherry. Old tuberculous process in the right apex of lung.

Remarks.—It has been a common observation that when these traumatic cranial patients merge very quickly from the condition of shock into that of high intracranial pressure without the patient having gone through the signs, clinically, of medullary compression—such as a very slow pulse- and respiration-rate, etc.—that these patients are most serious ones in that the condition of advanced medullary edema may progress so rapidly that very little can be done for them. This is particularly true with fractures about the foramen magnum and subtentorially, and the presence then of a high temperature within 8 hours after the injury is frequently observed in such posterior basal fractures—as though the heat centers in the basal ganglia were irritated. It would seem in this patient that the operation had really been performed during the early stage of medullary edema, for although the intracranial pressure was high yet the pulse was at its lowest level, only 74, and therefore confirming the opinion that the patient advanced very rapidly from the condition of shock into that of medullary edema without exhibiting clinically the signs of medullary compression. A bilateral subtemporal decompression might have been performed upon this patient on account of the very high intracranial pressure ascertained at operation, and yet I feel that the end-result would have been the same in view of the autopsy findings in the subtentorial cavity about the medulla. There are no more serious cranial injuries than those located in this region.

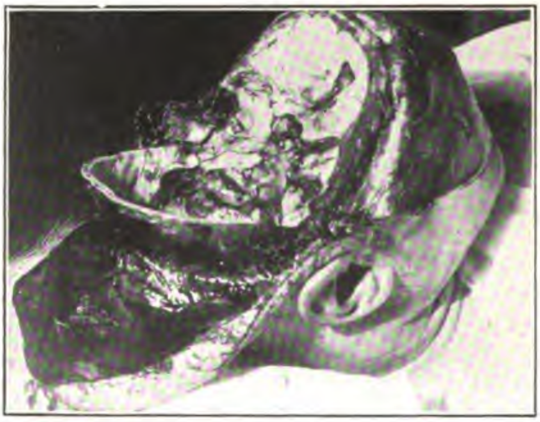


FIG. 99.—Photograph of the cranial fracture of the patient showing the extensive linear fracture of the left half of the occipital bone, extending into the petrous portion of the left temporal bone; the lower half of the bony opening of the left decompression with its drain of rubber tissue is clearly shown.

The rupture and operative laceration of the cortex beneath the Sylvian fissure upon enlarging the dural opening should have been prevented technically, and it now can be: if the dura is under very high tension so that upon incising it the underlying cortex might rupture as in this patient, a lumbar puncture is now performed and the cerebrospinal fluid is allowed to escape, and it is surprising how quickly the intradural pressure can usually be lowered so that it makes the opening of the dura a practically safe procedure and the underlying cortex naturally does not rupture. At times, through a very small dural incision, a ventricle puncture needle may be used to permit the escape of cerebrospinal fluid from the ventricles and in this manner the intradural pressure is lowered so that the dural opening may be safely enlarged; however, a ventricular puncture should only performed when absolutely necessary, and if a lumbar puncture is possi then the latter is preferable for this purpose of lowering the intradural pressure—the dura having been exposed and a small incision made in it. The danger of an acute medullary compression in the foramen magnum by the withdrawal of cerebrospinal fluid at lumbar puncture is practically nil when a small dural incision has been made previously, as in the subtemporal decompression. The ventricular puncture through a decompression opening necessitates the piercing of the cerebral cortex and subcortically in order to reach the ventricle, and it must cause a certain amount of cerebral damage, even though it is not demonstrated clinically; I believe that such punctures, therefore, should only be performed when absolutely necessary.

Case 73.—Acute severe brain injury associated with high intracranial pressure due to extradural, subdural and cortical hemorrages. Left subtemporal decompression and drainage. Death. Autopsy.

No. 274.—George. Forty years. White. Married. Manager. Scotland. Admitted June 4, 1915, Polyclinic Hospital.

Operation (June 5, 1915—19 hours after injury).—Left subtemporal decompression and drainage.

Died June 8, 1915—78 hours after operation.

Family history negative.

Personal history negative.

Present Illness.—Patient was found lying unconscious upon the curbing. As there was an odor of alcohol upon his breath, he was arrested for alcoholism and was confined in a cell at the police-station for 6 hours; not becoming conscious, he was transferred to the hospital in the ambulance.

Examination upon admission (7 hours after injury).—Temperature, 99.2°; pulse, 86; respiration, 20; blood-pressure, 110. Unconscious, but can be aroused by the fumes of ammonia; cannot talk. Alcoholic facies; the odor of alcohol still upon his breath. Hematoma over left parieto-occipital area. Bleeding from left ear and nose; extensive left mastoid ecchymosis. Pupils moderately dilated, equal and react normally. Reflexes—patellar present and equal; no ankle clonus nor Babinski; abdominal reflexes not elicited. Fundi—retinal veins slightly enlarged; no definite blurring of the margins of optic disks, but a general haziness throughout both retinæ.

Treatment.—Expectant palliative; routine shock measures instituted. The low blood-pressure associated with no marked signs of intracranial pressure indicated a mild degree of shock being present and the patient was treated expectantly in the belief that a good recovery could thus be obtained. Within 6 hours, however, the pulse descended to 60, blood-pressure rose to 130, and a definite weakness of the right side of face and right arm appeared with an incomplete motor aphasia. (Patient was ascertained to be right-handed as were his parents, brothers and sisters.)

Examination (11 hours after admission and 18 hours after injury).—Temperature, 100.2°; pulse, 48–50; respiration, 14; blood-pressure, 150. Profound unconsciousness. Discharge of blood and cerebrospinal fluid from the left ear had ceased 6 hours before; otoscopic examination reveals a laceration of the posterior quadrant of the left tympanic membrane. Marked weakness of right side of face (cortical in type) and of right arm, though no paresis of right leg could be elicited. Pupils—left much larger than right and does not react to light. Reflexes: patellar—right greater than left; right exhaustible ankle clonus and suggestive right Babinski; abdominal reflexes absent. Fundi—retinal veins slightly enlarged; no definite blurring of margins of optic disks but a general retinal haziness throughout and not limited to the optic disks. Lumbar puncture—blood-tinged cerebrospinal fluid under slightly increased intracranial pressure (14 mm.).

Treatment.—The clinical picture was very confusing and puzzling in that a patient, having a head injury, whose pulse was as low as 40 and respirations 14, with a blood-pressure rising from 110 to 150, indicating a high intracranial pressure and still increasing, and yet the ophthalmoscopic examinations were practically negative and the pressure of the cerebrospinal fluid as estimated at lumbar puncture was only moderately increased. For fear a medullary edema might be precipitated unless an immediate decompression was performed, this operation was considered advisable, and upon the left side on account of the mild motor aphasia, paresis of right side of face and of the right arm, increased right reflexes and a dilated left pupil.

Operation (12 hours after admission and 19 hours after injury).—(Pulse, however, was now 66 and respiration 18 and rather weak and shallow, so that medullary edema was feared.) Left subtemporal decompression (no anesthesia being necessary): usual vertical incision, bone removed and no complications; squamous portion of temporal bone contained several irregular lines of fracture running antero-posteriorly and vertically; the posterior portion of the fractured bone was depressed downward toward the petrous portion of temporal bone. Small extradural clot evacuated. Dura tense and bluish, and upon incising it dark bloody cerebrospinal fluid spurted a distance of 1 inch, and upon enlarging the dural opening many dark syrupy clots oozed out. Cortex very much congested throughout with many punctate hemorrhages. At lower anterior portion of the left temporal lobe, there was a laceration about 2 cm. in length. As the cerebral tension was very high and there was not a large amount of cerebrospinal fluid escaping, an effort was made to tap the ventricle through the lacerated cortical area but it was unsuccessful; however, by the end of the operation sufficient cerebrospinal fluid had escaped to permit the cerebral cortex to become less tense and to pulsate. Usual closure with 2 drains of rubber tissue inserted. Duration, 45 minutes.

Post-operative Notes.—At the end of the operation, the temperature was 101.4°, pulse 98, respiration 22, blood-pressure 138; at the time, I felt that medullary edema had not occurred but that the rise in the pulse- and respiration-rates and the lessening of the blood-pressure were merely due to the operation itself. However, the general condition of the patient gradually became worse, so that 20 hours after operation the temperature was 103.6°, pulse 116, respiration 32, blood-pressure 120, and this condition rapidly progressed—a typical medullary edema and collapse; at 42 hours after operation, the temperature was 106.4°, pulse 154, respiration 44, blood-pressure 106, and yet owing to the excellent resistance of the patient he did not die until 78 hours after operation.

Autopsy.—Irregular line of fracture from left vertex down into decompression area and then into left petrous bone to the internal auditory meatus and then backward along the basilar process into the anterior margins of the foramen magnum (Fig. 100). Thin layer of subdural blood over both hemispheres, and subtentorially about the medulla were much dark blood and bloody cerebrospinal fluid exerting direct compression upon the medulla itself. Punctate hemorrhages throughout the anterior portion of cortex of left hemisphere and a small laceration in the posterior portion of the third left frontal convolution (Broca's motor speech area). Ventricles negative.

Remarks.—This case was very instructive; a man considered as an alcoholic merely because he is unconscious and there is an odor of alcohol upon his breath, is first taken to the police station, then removed to the hospital where careful examinations reveal no signs of a definite increase of intracranial pressure and yet the pulse and respiration gradually became lower and lower and then, when the pulse does reach 48 and the respirations 14, it is considered necessary to perform a decompression even though the ophthalmoscopic examination reveals only slight increase and the lumbar puncture only a moderate increase of the intracranial pressure. The operation was not performed until the signs of an early medullary edema had already appeared and the patient died, as all these patients do, when medullary edema occurs after the pulse has reached its lowest level of medullary compression.

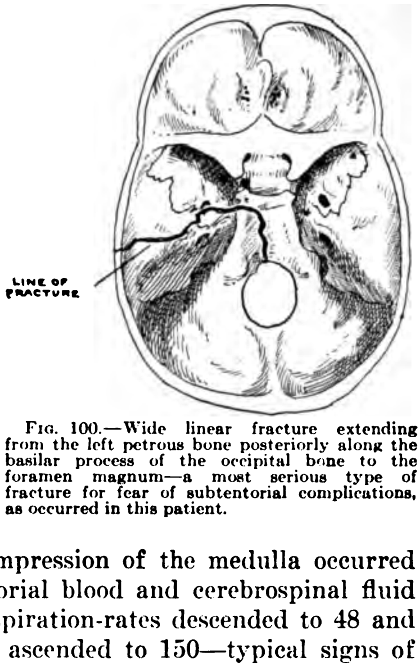


FIG. 100.—Wide linear fracture extending from the left petrous bone posteriorly along the basilar process of the occipital bone to the foramen magnum—a most serious type of fracture for fear of subtentorial complications, as occurred in this patient.

The autopsy findings "clear up" the puzzling clinical signs which seemed paradoxical. The patient upon admission to the hospital was in a mild condition of shock, as evidenced by the blood-pressure of 110 and the pulse-rate of 86; then, as the direct compression of the medulla occurred from the increasing amount of subtentorial blood and cerebrospinal fluid being collected there, the pulse- and respiration-rates descended to 48 and 14, respectively, and the blood-pressure ascended to 150—typical signs of medullary compression, but the ophthalmoscopic examinations were practically negative because the supratentorial pressure was not so greatly increased and the pressure of the cerebrospinal fluid as indicated by the lumbar puncture was also not markedly increased (as the low pulse- and respiration-rates would presuppose) because there was apparently a definite blockage at the foramen magnum, which prevented much cerebrospinal fluid and blood from escaping downward into the spinal canal, or the general intracranial pressure was in reality not high and there was only a high local medullary pressure.

A suboccipital decompression, if performed earlier than the time of the subtemporal decompression, might have relieved the acute medullary compression sufficiently to have secured a recovery; the presence of the motor aphasia and weakness of the right side of the face and of the right arm made the left subtemporal decompression seem at the time the more rational procedure.

The resistance of patients to intracranial injuries, and particularly to the effect of an increased intracranial pressure, varies very much indeed: children withstand them much better than adults, women apparently much better than men, and naturally non-alcoholic patients much better than those patients addicted to the daily use of alcohol; these latter patients are particularly bad risks and the prognosis, with or without operation, is always very grave, in that the danger of the so-called "wet" brain of cerebral edema, and consequently a medullary edema, is very great indeed. It is for this reason, to a large extent, that the mortality figures of large "city charity" hospitals are very high indeed, because the vast majority of their patients having brain injuries are also suffering from the results of chronic alcoholism and therefore arteriosclerosis in its various forms, chronic nephritis, etc.

If this patient could have been operated upon within two hours after admission, he might have had a definite chance of recovery, and it is unfortunate that the operation, when it was decided upon, could not have been performed within three hours after admission instead of five hours. At the first examination upon admission, the condition of the patient was such that it was felt advisable to examine him frequently in the hope that he would be able to "take care of" the increased pressure by the natural means of absorption, and this decision was considered advisable and good surgical judgment (as it is in many cases). In this patient, however, the signs of medullary compression advanced so rapidly that it would have been better judgment to have advised the operation earlier, and yet it was impossible to ascertain this fact within two hours after his admission. Yet when it was finally decided that an operation was imperative, it was most unfortunate that the operation could not have been performed immediately, for by the time the operation was finally begun the patient was well advanced into the condition of cerebral edema. I have observed this loss of most valuable and most important time to the patient in similar cases of brain injury when the patient has been allowed "to wait" over night in order that an X-ray picture of the fracture may be obtained; so frequently, a patient having the condition of mild medullary compression is thus permitted to reach the stage of medullary edema while waiting for an X-ray picture—of no importance either to the patient or to the doctor, so far as the treatment of the patient is concerned, as an operation is not advised according to the presence or not of a fracture of the skull; it so frequently happens that the operation should be performed upon the side of the skull opposite to the fracture, and it is the clinical signs which determine whether the operation shall be a left or a right subtemporal decompression. In these urgent cases, if the X-ray must be taken (and I believe an X-ray picture should always be obtained if only for the clinical records and data), then by all means let it be taken several days after the necessary and appropriate treatment has been used—whether an operation or not; otherwise, it may happen, and I feel that it does frequently occur, that the X-ray picture of the patient has been obtained and the patient has died or has been allowed

ngerous stage of medullary edema while "waiting" for the
erative treatment.

Acute severe brain injury associated with high intracranial
to subdural hemorrhage and cerebral edema. Left subtem-
ression and drainage. Death. Autopsy.

Daniel. Forty-four years. White. Married. Salesman. U. S.
October 21, 1913, Polyclinic Hospital. Referred by Doctor
dge.

(October 22, 1913—21 hours after injury).—Left subtem-
ression and drainage.

ber 23, 1913—18 hours after operation.

tory negative.

istory negative, except for chronic alcoholism.

lness.—While crossing the street in an intoxicated condition,
nocked down by a trolley car; immediate loss of consciousness;
hospital in the ambulance.

ion upon admission (40 minutes after injury).—Tempera-
pulse, 82; respiration, 20; blood-pressure, 146. Profoundly
alcoholic and vomited profusely alcoholic mixtures three times
lightly aroused. Extensive laceration of scalp over occipital
ecchymosis of both orbits. Blood in the right external auditory
pic examination reveals a laceration of posterior portion of
ic membrane; left tympanic membrane bluish and bulging;
areas ecchymosed. Pupils equal, rather contracted and do not
Reflexes negative. Fundi negative.

.—Expectant palliative.

ion (12 hours after admission).—Temperature, 99.8°; pulse,
n, 16; blood-pressure, 150. At this examination, careful prob-
ipital laceration reveals a fracture of the underlying bone, but
l. Patient has just had a Jacksonian convulsion, beginning
arm, then extending both to the right face and right leg;
of the muscles lasted 3 minutes. Pupils—left much smaller
d does not react to light. Reflexes: patellar—right greater
ht inexhaustible ankle clonus and double Babinski; abdominal
t. Fundi—retinal veins dilated; definite blurring of nasal
th optic disks. Lumbar puncture—bloody cerebrospinal fluid
ressure (approximately 21 mm.).

.—The onset of right-sided convulsions with the signs of an
tracranial pressure made a subtemporal decompression advis-
to lessen the intracranial pressure but also to lessen the cortical
the left hemisphere; on account of the right-sided convulsions,
right reflexes and the contracted left pupil, a left subtemporal
was naturally advised. Unfortunately, permission for the
ld not be obtained immediately, and when it was finally secured,
room was being used so that it was 8 hours later before the
ld be performed; during this time, the patient had had 14
nvulsions (none of them becoming general in character and
ot over 3 minutes), the pulse had descended to 58 three hours

before and had now risen to 76, both optic disks were obscured by edema but no measurable swelling of them, and the left pupil which had been contracted was now markedly dilated—so that the danger of medullary edema was very great indeed (and as the history of the patient shows, it had already begun).

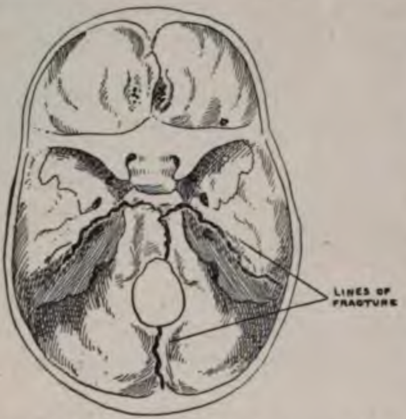


FIG. 101.—Extensive linear fractures of both petrous bones and of the occipital bone into the foramen magnum, causing a large subtentorial hemorrhage and direct medullary compression and its resulting medullary edema.

Operation (20 hours after admission).—Left subtemporal decompression (no anesthesia being necessary): usual vertical incision, bone removed and no complications. Dura very tense and bluish; upon incising it, dark syrupy blood welled through opening and upon enlarging the dural opening, much of this free subdural blood and bloody cerebrospinal fluid escaped. Cortex was under such high pressure and protruded so tensely that two attempts were made to tap the ventricles but they were unsucessful. Cortex contained many punctate hemorrhages but no lacerations were visible. During the operation, two right-sided convulsions occurred so that it was necessary to use chloroform to control them. At end of the operation, the cortex could be seen pulsating feebly. Usual closure with two drains of rubber tissue. Duration, 50 minutes.

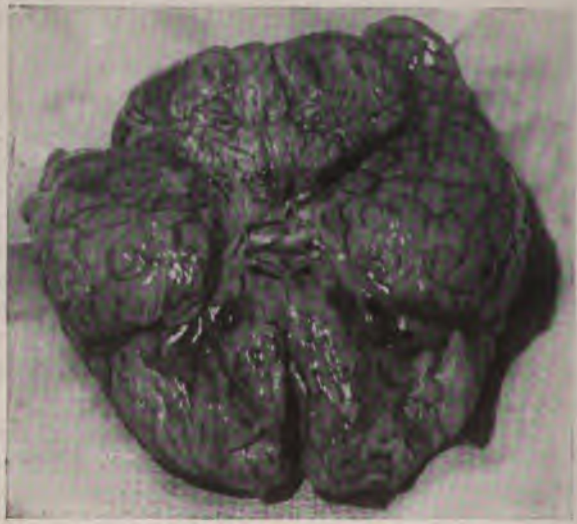


FIG. 102—Multiple subarachnoid hemorrhages especially upon the orbital surfaces of both frontal lobes in a patient dying from acute medullary edema.

Post-operative Notes.—The temperature at the beginning of the operation had been 102.6° and the pulse 92, and at the end of the operation it had risen to 104.4° and the pulse to 118; the general condition rapidly became worse, so that 12 hours after operation the temperature was 106°, pulse 150, respirations 42, whereas the blood-pressure was only 102—that is, all the signs of an acute medullary edema and collapse. Patient died 18 hours after the operation with a temperature of 108°.

line of fracture radiated from external occipital
l in median line across the foramen magnum and
ilar process of occipital bone to the posterior clinoid
ca; from this point a line of fracture radiated later-
ne (Fig. 101). Small hemorrhage beneath dura over
o posterior to both orbits. Much subdural free blood
and clotted blood subtentorially about cerebellum
ensive lacerations and cortical hemorrhage in both
the orbital surfaces (Fig. 102). Both middle ears

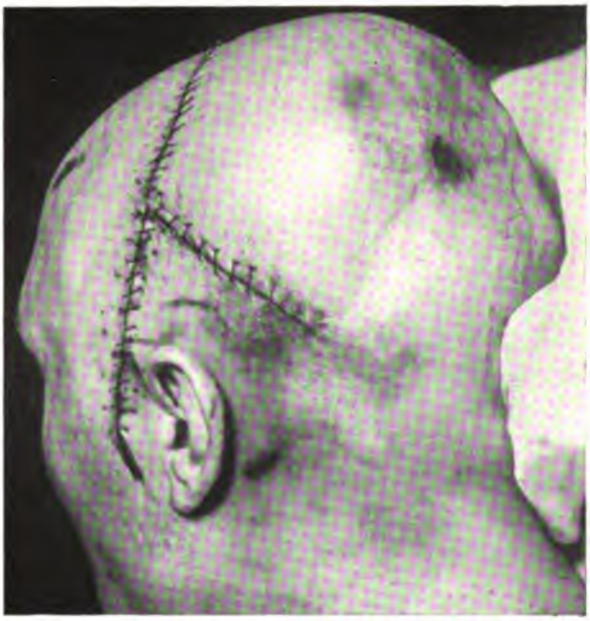


FIG. 103.—The post-mortem photograph after completion of autopsy exhibiting the multiple contusions of the scalp and the extensive mastoid ecchymosis.

emorrhage, acutely compressing the medulla itself.
g cause was found to account for the right-sided
x of both hemispheres appeared macroscopically to
ese negative findings to account for localized con-
s of focal cortical irritation are unfortunately only
njuries—whether due to localized edema as seems
ases, or that certain areas of the cortex are more
in some people than in others, may also be a
ce. It is, however, not so essential to attempt to
self from the particular area of the cortex affected
neral intracranial pressure by means of a subtem-
pecially over the hemisphere involved, and by this
n is also lessened.

ng in this patient to observe the pupillary changes;
slightly contracted and equally so, due to the mild

irritative presence of the cortical hemorrhage. (Shock not being an important factor in this patient, the pupils were not dilated as they usually are; alcoholism undoubtedly tends to lessen the initial shock in head injuries.) As the cortical irritation increased, and especially over the left hemisphere more than the right, the left pupil became more contracted than the right pupil; however, as the cortical irritation over the left hemisphere became overshadowed by the pressure of the increasing supracortical hemorrhage, the signs of cortical irritation changed to those of a cortical paralysis of pressure—that is, the contracted left pupil now became dilated and much larger than the right pupil, and naturally with no reaction to light. This is a fairly constant pupillary phenomenon and it is a definite aid to cortical localization, especially in the absence of external factors such as morphia, etc.

The otoscopic findings of a lacerated right tympanic membrane and merely a bluish and bulging left tympanic membrane were confirmed at the autopsy in that a line of fracture extended through the tegmen tympani of both petrous bones and yet the left tympanic membrane escaped being ruptured. It is surprising that no cerebrospinal fluid was observed in the discharge from the right ear, and yet that is a fairly frequent observation even in the presence of a line of fracture into the middle ear and the profuse discharge of blood from the external auditory canal; it would seem that in some of these patients, the dura overlying the line of fracture in the petrous bone is not torn and therefore there is no escape of cerebrospinal fluid.

CASE 75.—Acute severe brain injury associated with high intracranial pressure due to subdural hemorrhage and cerebral edema. Right subtemporal decompression and drainage. Death. Autopsy.

No. 430.—Frank. Thirty-eight years. White. Single. Thug. U. S.

Admitted November 19, 1915, Polyclinic Hospital.

Operation (November 20, 1915—26 hours after injury).—Right subtemporal decompression and drainage.

Died November 21, 1915—32 hours after operation.

Family history negative.

Personal History.—"Strong-arm" man with a bad reputation; twice in Sing Sing.

Present Illness.—Patient was found in an alley-way following a fight between rival "gangs"; unconscious from a head injury due to blackjack; brought to hospital in the ambulance.

Examination upon admission (about 2 hours after injury).—Temperature, 99°; pulse, 58; respiration, 16; blood-pressure, 118. Well-developed and nourished. Unconscious. Bleeding from the right ear and from the nose; no mastoid ecchymosis. A very superficial laceration of the scalp in the median occipital region. Pupils—very much contracted and no reaction to light (morphine believed to be the cause). Reflexes—both legs very rigid and it was impossible to elicit either patellar reflex; double exhaustible ankle clonus; right Babinski with a suggestive left Babinski; abdominal reflexes absent. Fundi—retinal vessels enlarged; nasal margins of both optic disks blurred. Lumbar puncture—blood-tinged cerebrospinal fluid under a moderately increased pressure (approximately 14 mm.).

Treatment.—Expectant palliative; careful observation. It was hoped that the increased pressure, as shown in the lowered pulse-rate and by the ophthalmoscopic examination and the lumbar puncture, could be "taken care of" by the normal means of absorption; the lowered blood-pressure of 118, due most probably to the initial shock of the head injury, was a definite contra-indication for operation. During the next 8 hours, the pulse slowly increased as high as 76 and the general condition of the patient improved—the blood-pressure rising to 130; at this time, however, the profuse bleeding from right ear and nose stopped, and within 2 hours the pulse gradually descended, while the blood-pressure continued to increase until the following examination was made.

Examination (22 hours after admission).—Temperature, 102°; pulse, 50; respiration, 14; blood-pressure, 142. Profound unconsciousness. Bleeding from the right ear has ceased; otoscopic examination reveals a small laceration of the posterior lower quadrant of the right tympanic membrane. Pupils—right widely dilated and does not react to light. Reflexes—patellar greatly exaggerated with double patellar clonus (both legs being very rigid and could not be flexed); double ankle clonus and double Babinski; abdominal reflexes absent. Fundi—retinal veins dilated; papilledema of 1 diopter—both nasal halves and temporal halves being obscured. Lumbar puncture—blood-tinged cerebrospinal fluid under high pressure (approximately 22 mm.).

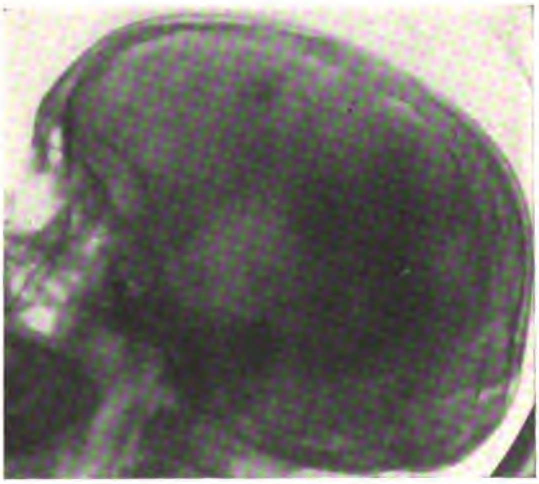


FIG. 104.—Extensive wide linear fracture of occipital bone just to the right of the midline, in a patient upon whom the operation of right subtemporal decompression was performed in the hope that an acute medullary edema could be prevented Two silver clips can be seen within the oval bony defect of the right subtemporal decompression.

Treatment.—An immediate right subtemporal decompression advised in the hope that the acute medullary compression could be relieved before the signs of medullary edema occurred and thus this great danger be avoided; the dilated right pupil, together with the fact that the patient and his relatives all were right-handed, and there being no other localizing signs, a right rather than a left subtemporal decompression was deemed advisable; the line of fracture extending into the right ear was of no localizing aid to the intracranial lesion. However, before preparations for the operation could be completed, the pulse, within a period of two hours, had risen rapidly to 76, while the blood-pressure had descended to 124; nevertheless, in spite of these signs of an approaching medullary edema, the operation was hurriedly performed.

Operation (24 hours after admission).—Right subtemporal decompression (no anesthesia being necessary): usual vertical incision, bone removed, and no complications. Dura tense and bluish, and upon incising it, a large amount of dark syrupy subdural blood welled out; upon enlarging the dural opening, many dark subdural clots extruded, revealing a swollen cortex which tended to protrude but did not rupture; rather "dry" brain, but owing to the loss of a large amount of supracortical free blood, the cortex could be seen pulsating slightly at the end of the operation. No punctate hemorrhages nor cortical lacerations visible. Usual closure with two drains of rubber tissue inserted. Duration, 35 minutes.

Post-operative Notes.—The general condition of the patient did not become worse during the first 12 hours after operation—the pulse remained under 80 and the blood-pressure not lower than 120, but then the condition of the patient rapidly changed so that 20 hours after operation the temperature was 105.4°, pulse 132, respiration 34 and blood-pressure 110—a pronounced condition of medullary edema. The usual course followed—temperature rising to 108°, pulse 160 plus, respirations 48, while the blood-pressure descended to 80 and below, when death occurred 32 hours after operation. A röntgenogram was taken postmortem for comparison with the autopsy findings (Fig. 104).

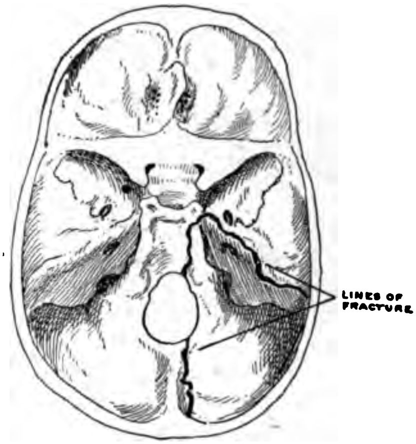


Fig. 105.—Extensive linear fracture of right petrous bone and of the occipital bone extending into foramen magnum anteriorly and posteriorly—the most serious type of fracture on account of the frequent medullary complications of direct pressure of hemorrhage and edema.

Autopsy.—Linear fracture extended downward from occipital protuberance into posterior margin of foramen magnum and then from the anterior margin of foramen magnum forward along the right edge of the basilar process to the tip of the right petrous bone, where it turned at right angles into petrous portion of right temporal bone (Fig. 105). Blood in the right middle ear. Layer of dark blood over both hemispheres—more over left. Both anterior portions of frontal lobes contused and slightly lacerated—left more than right; many subarachnoid hemorrhages (Fig. 106). Much clotted blood subtentorially about the cerebellum and medulla—the medulla being forced downward into the foramen magnum where it apparently blocked the entire lumen. Ventricles negative.

Remarks.—It would seem that a suboccipital decompression would have offered this patient a greater chance of recovery if performed earlier—before the pulse had reached its lowest level and therefore before the signs of a beginning medullary edema had appeared; this operation however, is a much more formidable one than the subtemporal decompression, and the signs of an acute direct medullary compression occur so rapidly and progress so quickly into the stage of medullary edema that it is most unusual for a suboccipital decompression to be deemed advisable; besides, even in these patients having an acute direct medullary compression but of mild severity, the subtemporal decompression undoubtedly suffices to obtain a good recovery of the patient, and as the subtemporal decompression is a comparatively "safe" operation, and of very little shock to the patient, naturally it is usually advised. When not successful and the patient dies and similar autopsy findings as in this patient are disclosed, then it is very easy to assert that a suboccipital decompression was indicated and would have proved sufficient for a recovery of life. If a subtemporal decompression with drainage and, if necessary, a bilateral decompression had been performed 6 hours earlier, when the pulse was descending from 76 to 60 and the signs of an increasing intracranial pressure were becoming more marked, rather than waiting until morning when the pulse had descended to 50 (its lowest level as it proved), possibly even in this patient a recovery might have been obtained, as the subtentorial pressure and even the direct compression of the medulla might have been lessened. There are no cases in surgery requiring immediate treatment so much as patients having brain injuries with a high increase of the intracranial pressure, and the successful treatment of ones having a high intracranial pressure requires most careful and repeated examinations and observations, and if the increased pressure cannot be controlled and lessened by the expectant palliative measures, then an early mechanical relief by means of a decompression is urgently required and should be performed without delay.

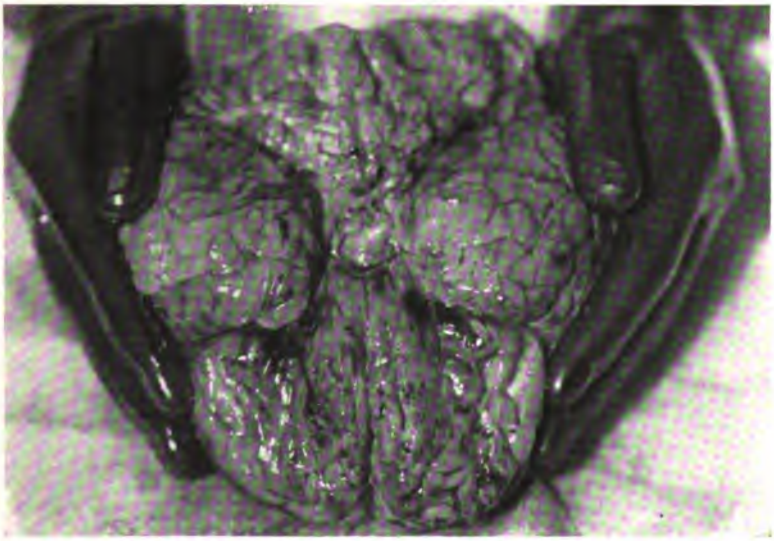


FIG. 106.—Multiple subarachnoid hemorrhages upon the inferior surfaces of both frontal lobes in a patient dying of acute medullary edema following a medullary compression of subtentorial hemorrhage.

The unusually low initial pulse-rate of 58 in this patient, while the blood-pressure was only 118, would tend to indicate even then a direct medullary compression while the blood-pressure itself was influenced, temporarily at least, by the initial shock. In future patients, this initial low pulse combined with a low blood-pressure will have a greater significance.

The markedly contracted pupils observed upon admission were undoubtedly due to the cortical irritation of the subdural hemorrhage, and as the intracranial pressure became higher and more over the right cortical hemisphere, the right pupil naturally became dilated from the paralytic effect of this supracortical pressure.

The spasticity of both legs to the extent of a marked stiffness, and also slightly of the arms, is a very serious prognostic sign in these patients having brain injuries in that they usually die within 24 hours after its appearance; it may be due to extreme intracranial pressure, but it is more probably the result of high intracranial pressure causing an edema of the pyramidal tracts and particularly of the medulla itself, so that this extreme degree of bilateral spasticity occurs. The autopsy findings of the medulla being "choked" and "collared" in the foramen magnum would tend to confirm this observation; also the presence of a subtentorial fracture and thus the greater danger of subtentorial lesions with high direct medullary compression, is usually ascertained in these patients having a high degree of bilateral spasticity.

The "*contre-coup*" effect of cranial injuries upon the brain is again illustrated in this patient—the area of contact being in the occipital region and the fracture extending from there along the base to the right ear, and yet it is not the underlying or overlying cortex that is damaged but the anterior portions of both frontal lobes, and particularly the left lobe, which are contused and lacerated. However, it is the presence of an increased intracranial pressure which determines the necessity of a subtemporal decompression, and not the presence or not of cortical contusions and laceration; also, it is not necessary nor practicable in most patients having brain injuries to operate directly over the cortical lesion, but rather to relieve the high intracranial pressure by means of a subtemporal decompression over the hemisphere of the higher pressure—whether the underlying cortex is contused or lacerated or not, and it matters not where the linear fracture of the skull is or whether it is present at all; if there are no localizing signs of greater increased intracranial pressure over either hemisphere, then naturally the subtemporal decompression is performed on the right side in patients who are right-handed, and on the left side in the patients who are left-handed.

The accurate estimation of the increasing intracranial pressure, as registered by the lumbar puncture, is clearly illustrated by the test made upon the patient's admission to the hospital when the pressure was approximately 14 mm., and then 22 hours later when the signs of definite papilledema had occurred in both fundi, the pressure at lumbar puncture had risen to approximately 22 mm. This test is now more accurately registered by the use of the spinal mercurial manometer and this instrument is a most valuable one for the accurate registration of increased intracranial pressure of varying degree.

Case 76.—Acute severe brain injury associated with a fracture of the base of the skull and extreme intracranial pressure; extradural, subdural and intracerebral hemorrhages and cortical lacerations; medullary compression and incipient medullary edema. Left subtemporal decompressi and drainage. Medullary edema; death. Autopsy.

No. 1026.—Gertrude. Fifteen years. White. School. U. S

Admitted October 29, 1918—5 hours after injury. Audubon Hospital. Referred by Doctor George Barrie.

Operation (October 29, 1918—1 hour after admission).—Left subtemporal decompression and drainage.

Died October 30, 1918—7 hours after operation.

Family history negative.

Personal history negative.

Present Illness.—While "running an errand" early this evening (7 P.M.), patient was found lying upon the pavement near the curbing in a stuporous condition; she was carried into her home, regained complete consciousness almost immediately, said that her head "hurt" her, but was unable to state how the injury had occurred; small amount of bleeding from left ear, associated with a left mastoid ecchymosis; she became sleepy—"dozed off to sleep," and the patient was not considered to be seriously injured until 3 hours later, when it was ascertained that she could not be aroused to consciousness; the pulse had become 66 and the respirations 14 and of an irregular character, while convulsive twitchings of the entire right side of the body now appeared.

Examination (3 hours after injury—in consultation with Doctor Barrie). —Temperature, 99.4°; pulse, 64; respiration, 16; blood-pressure, 128. Well-developed and nourished. Profound unconsciousness with extreme spasticity of both legs and partially of both arms—almost the rigidity of opisthotonus; tetanic twitches throughout body—right side more than left. Small amount of clotted blood in left auditory canal; otoscopic examination revealed a laceration of the lower posterior quadrant of the left tympanic membrane; extensive left mastoid ecchymosis. Pupils—left pupil widely dilated, while the right pupil was contracted to pin-point size, and neither reacts to light. Reflexes—patellar very much exaggerated, with double patellar clonus; double ankle clonus and double Babinski; abdominal reflexes absent. Fundi—retinal veins full, tortuous and buried in edematous tissue in places; double papilledema of 1 diopter swelling—both nasal and temporal halves of the optic disks being obscured by edema but not to the extent of producing "choked disks" (a swelling of 2 diopters plus).

Treatment.—In the presence of these signs of high intracranial pressure of sufficient amount to produce the definite condition of medullary compression associated with a bilateral spasticity and epileptiform seizures of cortical irritation, an immediate removal of the patient to a hospital was advised so that the patient could have the benefit of a subtemporal decompression if her condition then warranted it—the danger of an acute medullary edema being very great indeed.

Examination upon admission to Audubon Hospital (4 hours after injury).—Temperature, 101°; pulse, 88; respiration, 28; blood-pressure, 126. Profound unconsciousness continues; the spasticity is possibly not so marked and no convulsive seizures. Pupils—both dilated and do not react to light. Reflexes—patellar very much exaggerated with double patellar clonus; double ankle clonus and double Babinski; abdominal reflexes cannot be elicited. Fundi—retinal veins engorged and buried in places in edematous retinæ; double "choked disks" of 3 diopters swelling. Lumbar puncture—bloody cerebrospinal fluid under high pressure (23 mm.).

Treatment.—For fear of the onset of an acute medullary edema, an immediate left subtemporal decompression and drainage advised—the relatives being informed that the patient's chances of recovery were very small but that the operation to lessen the high intracranial pressure did offer some hope and that it was the only form of treatment which might be beneficial.

Operation (5 hours after injury).—Left subtemporal decompression and drainage (before the operation could be started, however, the pulse had ascended to 110 and the respiration to 34, while the blood-pressure had descended to 120—the signs indicative of an early medullary edema; it was still hoped that the immediate relief of the high intracranial pressure might aid the patient to recover, and it was therefore hurriedly performed): usual vertical incision, bone removed and no complications; temporal muscle beneath the temporal fascia was hemorrhagic and therefore a fracture of the underlying bone was revealed upon retraction of the muscle fibres; this fracture extended transversely through the upper portion of the left squamous bone and through it was oozing much free blood; upon removing the bone, a large extradural hemorrhage was revealed and evacuated. At the lower anterior angle of the bony opening, the underlying dura was found to be torn—also the main branch of the left middle meningeal artery which was spurting freely; a small gauze tape was used to control this bleeding temporarily. Through the dural opening much dark currant-jelly blood was being extruded, together with macerated cerebral tissue; the dura was now widely opened, exposing much subdural blood, and through several cortical lacerations welled dark blood-clots. The escape of this free hemorrhage and cerebrospinal fluid permitted the cortex to bulge less tensely so that a definite pulsation was visible. Usual closure with 3 drains of rubber tissue inserted. Duration, 38 minutes.

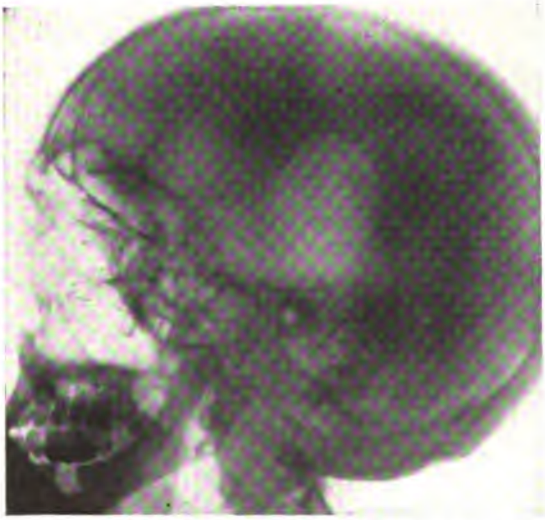


FIG. 107.—Oval bony defect of left subtemporal decompression in a patient dying from an acute medullary edema. Operation advised in the hope that the advancing medullary edema could be checked.

Post-operative Notes.—At the end of the operation, the temperature was 102°, pulse 140, respiration 36, while the blood-pressure had descended to 112; the patient did not recover consciousness and 2 hours after operation, convulsive twitchings began again on the right side of the body; the restlessness was extreme. The general condition of the patient rapidly became worse in that the temperature ascended to 106°, pulse and respiration to 150 and 44, respectively, while the blood-pressure descended below 100—the typical chart of an acute medullary edema approaching the exitus; patient died 7 hours after operation. A röntgenogram was taken postmortem for comparison with the autopsy findings (Fig. 107).

Autopsy.—Linear fracture extended transversely from the left external orbital angular process backward through the left squamous bone and then downward through the left petrous bone across the sella turcica to the foramen ovale in the right middle fossa (Fig. 108). Small amount of extradural hemorrhage lay beyond the margins of the left subtemporal decompression. A thin film of supracortical hemorrhage over both cerebral hemispheres, with extensive lacerations of the left frontal lobe and left temporo-sphenoidal lobe, while the tip of the right temporo-sphenoidal lobe was contused (*"trauma au contre-coup"*). In the posterior portion of left frontal lobe was an intracerebral hemorrhage of 4 cm. in diameter. Both the cerebrum and cerebellum were very edematous and "water-logged" with much free blood beneath the tentorium; the medulla itself was distinctly edematous. Ventricles negative.

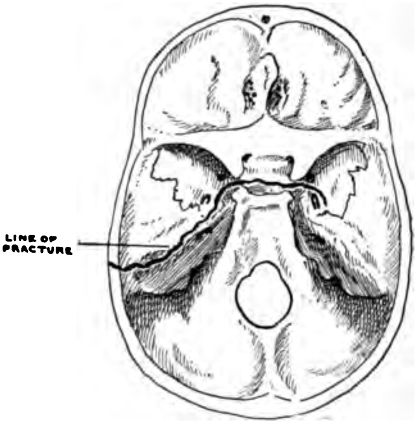


Fig. 108.—Extensive basilar fracture of the middle fossæ and across the sella turcica in a patient having large extradural, subdural and intracerebral hemorrhages and cortical lacerations—the resulting increased intracranial pressure producing an early medullary edema.

Remarks.—This patient again illustrates the futility of cranial operations upon patients who have reached the stage of medullary edema; it is useless to operate upon these patients after they have passed through the stage of medullary compression and have reached the condition of medullary edema, as indicated by the rapidly increasing pulse- and respiration-rates, high temperature and lowering of the blood-pressure. It would have been better surgical judgment to have refused to operate upon this patient after all preparations for the operation had been made and when it was ascertained that the condition of the patient had changed so rapidly from that of the preceding examination; and yet it is most difficult not to advise the operation even at this late stage, because it is definitely known that the patient will die if nothing is done, and that possibly the patient might have a chance of recovery if the operation is immediately performed before the medullary edema has advanced to a severe degree; this reasoning, however, is faulty and illogical, and it is poor surgical judgment to advise a cranial operation upon these patients after they have reached this stage of acute medullary edema.

The variation of the pupil in this patient is very interesting and instructive: the widely dilated left pupil associated with the contracted right pupil indicated the paralytic compressive effect over the left cerebral cortex and the irritative effect of a smaller supracortical hemorrhage over the right hemisphere; as this supracortical pressure over the right hemisphere increased, then the right pupil became dilated—indicating an extreme intracranial pressure of supracortical character. If this increased intracranial pressure could have been lessened by the operation to a marked extent, then the left pupil would undoubtedly have become smaller and thus indicating the subsidence of the paralytic effect of a left supracortical hemorrhage, and if a bilateral decompression had been performed, then the right pupil would also have become smaller; in this patient, however, the cerebral edema and intracranial hemorrhage were of such large amount and the condition of the patient had advanced so far into the condition of acute medullary edema, that the operation in itself was not of sufficient decompressive effect at this late stage to produce any marked improvement.

The bilateral spasticity observed within 3 hours after the injury was a bad prognostic sign, especially when so severe as to cause a mild degree of opisthotonos; it indicates not merely an extreme cortical irritation but also an edematous compression of the pyramidal tract fibers, and it is rare for these patients to recover; I have not had one patient in this series of acute brain injuries recover in whom this condition of marked bilateral spasticity has been observed.

The cessation of the convulsions at the time of the patient's admission to the hospital and also a lessening of the bilateral spasticity indicated that the cortical irritation had been submerged by an increasing supracortical pressure, so that the signs of cortical irritation, especially the convulsive twitchings, disappeared; in this connection, it is interesting to note that the convulsive twitchings returned to the right side of the body within 2 hours after the operation, when the pressure over the left cerebral cortex at least had been lessened, and it was thus possible for the cortical irritation to be again exhibited.

B. *Bilateral decompression.*

CASE 77.—Acute severe brain injury with signs of high intracranial pressure due to subdural hemorrhage and cerebral edema. Bilateral decompression and drainage. Medullary edema; death. Autopsy.

No. 191.—Dennis. Forty-eight years. White. Married. Laborer. Ireland.

Admitted October 30, 1914, Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Operations (October 30, 1914—5 hours after injury).—Bilateral decompression and drainage.

Died November 1, 1914—22 hours after operations.

Family history negative.

Personal history negative, except for chronic alcoholism.

Present Illness.—Three hours ago, patient fell headlong from doorstep—a distance of 5 feet, striking his head upon the cement pavement; immed loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (3 hours after injury).—Tempera 99.8°; pulse, 50; respiration, 14 (Cheyne-Stokes type); blood-pressure Profound unconsciousness. Contusion and laceration of scalp ove frontal area; left orbit ecchymotic and extensively swollen. Bleedin nose, mouth and left ear. Pupils—left larger than right and does n to light. Reflexes—patellar very much exaggerated but apparently equal; no ankle clonus nor Babinski; both cremasteric and abdominal reflexes absent. Fundi—dilated retinal veins; papilledema of both optic disks but having a measurable swelling of only one diopter—that is, not a fully developed "choked disk."

Treatment.—The patient being right-handed and the only localizing sign being a dilated left pupil (which tended to indicate that the left cerebral hemisphere was compressed more than the right), it was decided that a right subtemporal decompression was immediately advisable in order to lessen the high intracranial pressure and prevent the condition of acute medullary compression from merging into an acute medullary edema. While preparations for the operation were being completed, the pulse descended to 48 as its lowest level, but by the time the operation was begun, the pulse had ascended to 70, while the blood-pressure had fallen to 134—thus indicating even at this early date the probable onset of acute medullary edema; in the belief, however, that the patient might yet have a chance of recovery by means of an operation (an opinion now known to be mistaken), the operation was performed. A röntgenogram was made while waiting for the operating-room to be prepared (Fig. 109).

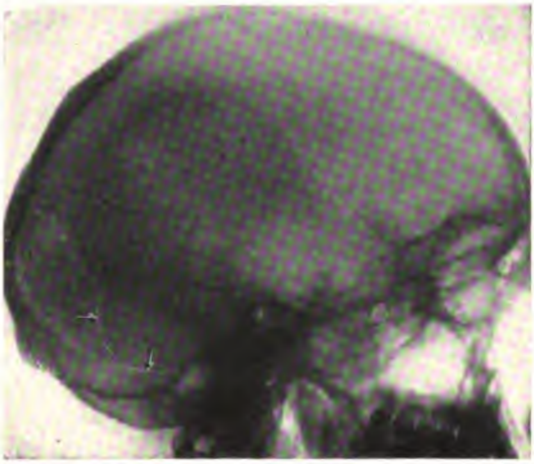


FIG. 109.—Curvilinear fracture of left occipital bone, extending into left petrous bone, in a patient having an extreme intracranial pressure precipitating a medullary edema. A bilateral subtemporal decompression was advised in the mistaken belief that a recovery of life was possible.

First Operation (2 hours after admission and 5 hours after injury).—Right subtemporal decompression; usual vertical incision, bone removed and no complications. Dura very tense and upon incising it, clear cerebrospinal fluid spurted under high pressure; upon enlarging dural opening, the edematous "wet" cortex protruded under high tension but did not rupture; no cortical hemorrhage nor laceration visible and the brain became comparatively "dry," but remained very much swollen and "water-logged." As only a moderate amount of cerebrospinal fluid had escaped and since the pulsation of the cortex was very slight, it was decided to perform immediately a left subtemporal decompression. Usual closure with 2 drains of rubber tissue inserted. Temporary sterile dressing applied.

Second Operation (immediately after first operation).—Left subtemporal decompression: usual vertical incision, bone removed, and no complications. Dura very tense and bluish; upon incising the dura, dark blood spurted a distance of 1 foot for a period of a minute; upon enlarging dural opening, large dark clots welled out so that the underlying cortex which was concave due to the compression of the overlying clots now became gradually convex. Dark clots, the size of cherries, continued to well into opening from above, though no cortical hemorrhage or laceration was visible in this area. At the end of operation, however, so much blood and cerebrospinal fluid had escaped that the intradural tension was much less and the cortex pulsated fairly normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 85 minutes.

Post-operative Notes.—Before the operation was finished, the pulse had ascended to 78 and, within 6 hours after operation, the pulse had risen to 110, respirations to 30, while the blood-pressure had descended to 104; the general condition of the patient rapidly became worse so that patient never regained consciousness; 16 hours after operation, the temperature was 106°, pulse 160, respiration 38, and the blood-pressure 90; the patient died 22 hours after operations.

Autopsy.—A curvilinear fracture extended through the left occipital bone forward and obliquely downward into the left squamous bone and then into the left petrous bone—ending near the left internal auditory meatus (Fig. 110). Only a small amount of subdural bood was present, but the cerebral cortex was very much swollen and hemorrhagic throughout. Much free blood and cerebrospinal fluid subtentorially about the cerebellum and medulla—this indicating a direct medullary compression. Ventricles negative.

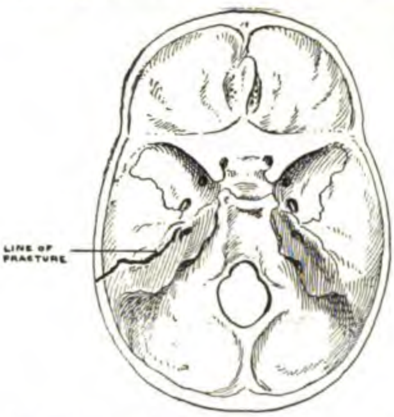


FIG. 110.—Linear fracture of left petrous bone in a patient dying from medullary edema due to the high pressure of a subdural hemorrhage and cerebral edema.

Remarks.—The early marked signs of high medullary compression occurring so quickly following a head injury indicated a direct medullary compression; for this reason, no lumbar puncture was attempted, although now it would be performed safely by using the spinal mercurial manometer so that there would be no danger of increasing the medullary compression by a "choking" of the medulla in the foramen magnum; also, if such a patient was now examined, a suboccipital decompression would be immediately performed rather than the subtemporal decompressions—which are of little or no value in relieving the acute direct medullary compression occurring in head injuries. It would have been better surgical judgment not to have performed the operation when it was ascertained that the signs of medullary edema had already occurred; if this patient could have been operated upon two hours earlier, a recovery of life might have been obtained.

Alcohol as a factor in increasing the mortality of brain injuries by lessening the natural resistance of the patient, so that medullary edema occurs much more easily in these patients, was present in this case; the mortality, following brain injuries in patients suffering from chronic alcoholism, is exceedingly high.

In the presence of very high intracranial pressure as in this patient, although having the localizing sign of a dilated left pupil—indicating the paralytic effect of higher pressure over the ipsolateral left cerebral hemisphere, it is better surgical judgment to perform first, a right subtemporal decompression in such a patient who is right-handed, and then the decompression over the side of the head having the signs of the higher pressure; in this manner, there is less danger of severe operative damage to the more highly developed underlying left cerebral cortex. Naturally, in patients having signs of only an increased intracranial pressure and not of extreme degree, then the decompression would be performed over the side of the head, exhibiting the signs of higher intracranial pressure—in this patient, the left hemisphere.

CASE 78.—Acute severe brain injury associated with signs of high intracranial pressure due to subdural hemorrhage and cerebral edema. Bilateral decompression and drainage. Medullary edema; death. Autopsy.

No. 789.—Daniel. Twenty-eight years. White. Single. Chauffeur. U. S. Admitted February 25, 1917, Polyclinic Hospital.

Operations (February 25, 1917—24 hours after injury).—Bilateral decompression and drainage.

Died February 25, 1917—immediately following operations.

Family history negative.

Personal history negative, except for chronic alcoholism.

Present Illness.—While patient was walking across the street, he was struck by an automobile and knocked headforemost into an iron water-hydrant; immediate loss of consciousness; brought to the hospital in the automobile.

Examination upon admission (15 minutes after injury).—Temperature, 98.4°; pulse, 60; respiration, 20; blood-pressure, 136. Well-developed and nourished; unconscious, but very restless. An extensive hematoma over the left parietal area extending down into left mastoid region. Profuse bleeding from left ear; left mastoid ecchymosis. Pupils—pin-point, equal and do not react to light. Reflexes—patellar very much exaggerated, right greater than left; right ankle clonus, right Babinski and a suggestive right Oppenheim and right Gordon reflex; abdominal reflexes absent. Fundi—unable to examine the fundi with the ophthalmoscope on account of the extreme contraction of both pupils.

Treatment.—Expectant palliative; careful observation. Within 1 hour, however, both pupils became widely dilated, right more than left, and did not react to light. Reflexes remained the same as at preceding examination, but the ophthalmoscope revealed the retinal veins markedly dilated with extensive retinal hemorrhages and an edematous obscuration of both optic disks—a papilledema of 1 diopter in swelling. Lumbar puncture—bloody cerebrospinal fluid under high pressure (21 mm.). The pulse had ascended to 92, while the respiration was irregular but deep, with a blood-pressure of 120, and the patient was becoming more and more profoundly unconscious.

Treatment.—In the belief that the pulse-rate of 92 was possibly due to the profuse hemorrhage from the left ear (120–130 drops per minute were escaping) and that this increase of the pulse-rate was not due to an early onset of medullary edema, it was thought advisable to perform a left subtemporal decompression immediately in the hope that this fatal complication might be avoided. While waiting for the operating-room to be prepared, a röntgenogram was taken, revealing an occipital fracture (Fig. 111).

First Operation (2 hours after admission).—Left subtemporal decompression (no anesthesia being necessary): usual vertical incision, bone removed and no complications. Dura exceedingly tense and bluish, and upon incising it, bloody cerebrospinal fluid spurted a distance of 3 feet, striking the operator in the eye; upon enlarging dural opening, the underlying cortex protruded and ruptured for a distance of 2 cm.; an attempt to tap the left lateral ventricle not successful, and as the underlying cortex was comparatively "dry" but "water-logged," and under such high tension that the cortex did not pulsate, a right subtemporal decompression was considered necessary. Usual closure with 2 drains of rubber tissue inserted. Temporary sterile gauze dressing applied.

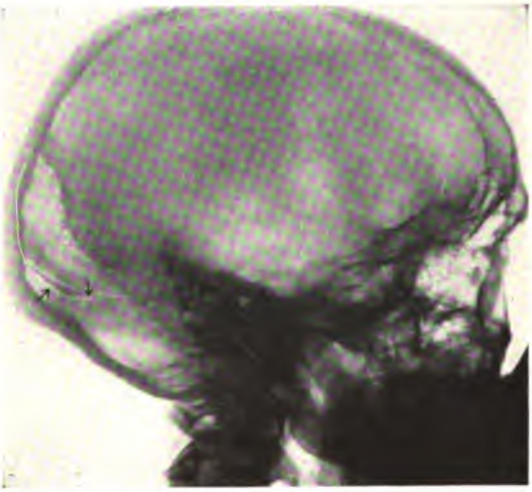


FIG. 111.—Linear fracture of the right half of occipital bone, extending into the right petrous bone in a patient upon whom a bilateral decompression was performed in the hope that it might prevent the advance of an early medullary edema.

Second Operation (immediately following the first operation).—Right subtemporal decompression: usual vertical incision, bone removed and no complications. Dura very tense and upon incising it, bloody cerebrospinal fluid spurted a distance of over 2 feet; upon enlarging dural opening, the underlying cortex tended to protrude but did not rupture. A small amount of cerebrospinal fluid and blood escaped, but not of sufficient quantity to permit the cortical tension to lessen. No cortical hemorrhage or laceration visible—merely a "water-logged" brain. Usual closure with 2 drains of rubber tissue inserted. Duration, 85 minutes. As the skin was being sutured, the patient suddenly became worse, pulse became imperceptible, respirations ceased, although the heart-beat continued for almost 3 minutes until the patient died—a death typical of medullary edema in that the heart-beat had ascended rapidly to 150 plus and the respirations to 40 plus, while the blood-pressure had descended to 84 before death.

Autopsy (Doctor Weston).—Large hematoma over left temporo-par occipital area. Wide linear fracture extended from posterior occipital tuberance downward and forward to the left of the foramen magnum then forward and to the left—tearing the left lateral sinus and then int left petrous bone along its superior margin toward the sella turcica several lines of fracture radiating anteriorly and laterally from it. (*See frontispiece.*) Large subtentorial hemorrhage about the medulla compressing it. Small amount of subdural hemorrhage over both hemispheres. Brain tissue itself not damaged, but very much swollen and "water-logged." Ventricles negative. The orbital surface of both frontal lobes slightly contused.

Remarks.—The question of shock in this patient was seriously considered as being an important factor in the condition, especially regarding the advisability or not of an early operation being attempted; the signs of a rapidly increasing pressure, however, with a blood-pressure of 136, made the factor of shock of almost negligible character and could not be considered as prohibiting an attempt to improve the condition of the patient. However, the increase of the pulse-rate from 60 to 92, while the blood-pressure decreased from 136 to 120, should have indicated to us that a medullary edema had already occurred and that the prognosis was absolutely bad—with or without operation—and naturally, no operation should have been performed.

The autopsy findings indicate such a high intracranial pressure, and especially such a direct compression of the medulla by subtentorial blood and cerebrospinal fluid, that a medullary edema seems inevitable. The rupture of the left sigmoid sinus is in itself sufficient cause of death in that when it bleeds subtentorially a direct compression of the medulla results.

The presence of chronic alcoholism in this patient was undoubtedly a factor in allowing the medullary edema to occur much more easily than in a patient of greater resistance, and yet any patient, no matter how resistant, would have succumbed to an injury of this severe character.

It would have been better judgment if a lumbar puncture with the measurement of the pressure of the cerebrospinal fluid had been made immediately after the patient's admission to the hospital, and it is always now performed in similar patients; so mild were the signs of shock at this time, that it could have been performed with little or no disturbance of the patient, and in this manner the accurate estimation of the intracranial pressure could have been obtained and possibly an earlier attempt to lessen the increased intracranial pressure would have been possible. And yet, the autopsy findings were of such a severe character in this particular patient that it is very doubtful whether any operative procedure could have benefited him.

A lumbar puncture was most carefully performed upon this patient before operation because it was believed that the subtentorial pressure was very high; although the patient was in the condition of shock, having a subnormal temperature and a low blood-pressure, yet the pulse was 60—a clinical syndrome in these patients indicating a high direct medullary compression in the presence of shock. To perform a lumbar puncture, therefore, and to allow the escape of cerebrospinal fluid might thus take away, to a greater or less degree, some of the supporting pressure beneath the medulla, and thus permit the medulla to be jammed down into the foramen magnum, producing the acute symptoms and signs of extreme medullary compression. This has occurred in patients having large subtentorial tumors, but since the lumbar puncture can now be so performed that no fluid is withdrawn, and its pressure can be estimated without lessening to any appreciable extent the intraspinal pressure, therefore the danger of this medullary complication is practically nil. In this patient, however, the subtentorial pressure was in itself so high that the same effect upon the medulla was produced, in that the medulla was "choked" in the ring of the foramen magnum. A suboccipital decompression should have been considered in this patient.

In the operations upon these patients, especially upon the right side during the first operation, a ventricular puncture should always be performed in the hope that the ventricles are dilated with fluid and thus the cerebral tension can be immediately lessened by the withdrawal of this fluid; this procedure should be attempted in all cases having extreme intracranial pressure. The autopsy findings, however, in this patient showed that the ventricles had not been blocked and therefore a ventricular tapping would have been of no assistance.

CASE 79.—Acute severe brain injury associated with a perforating bullet injury of entire brain and with high intracranial pressure due to subdural, intracerebral and ventricular hemorrhage. Bilateral decompression and drainage. Death. Autopsy.

No. 021.—Oliver. Sixty-six years. White. Married. Retired. U. S.

Admitted August 15, 1913—3 hours after injury, Polyclinic Hospital. Referred by Doctor W. S. Pritchard.

Operations (August 15, 1913—1 hour after admission).—Bilateral decompression and drainage.

Died August 16, 1913—12 hours after operation.

Family history negative.

Personal History.—Always well and strong; no alcoholism. Following the burning of his country home 3 years ago, patient became melancholy, depressed, and developed a large number of eccentricities: became very miserly and penurious—permitted meat to be used only once a week in the family in order to lessen the expense, and also prohibited the use of the bath-tub but once a week to save water and to prevent the "wearing-out of the pipes" by the running water; the use of the toilet was also included.

Present Illness.—Three hours ago (11 A.M.), the patient was found in the bathroom shot through the head—one .32-calibre revolver bullet passing through the vertex of the skull transversely and the other bullet entering at the vertex and perforating the brain vertically downward to lodge just posterior to the foramen magnum. Profuse bleeding from the vertex—being both the wounds of entrance and of exit of one of the bullets. Unconscious; brought to the hospital in the ambulance.

Examination upon admission (3 hours after injury).—Temperature, 99.2°; pulse, 74; respiration, 16; blood-pressure, 152. Well-developed and nourished. Profoundly unconscious with stertorous irregular respiration of the Cheyne-Stokes type. Profuse bleeding and discharge of bloody cerebrospinal fluid from the large bi-parietal wound at the vertex—4 inches in width and extending above the longitudinal sinus; small perforating d wound one inch to the right of the longitudinal sinus—the entrance other bullet. No definite paralyses elicited. Profuse discharge of blood and cerebrospinal fluid from the right ear. No powder burns or marks found. Pupils moderately dilated but equal, and react to light sluggishly. Reflexes —patellar present and equal; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi—retinal veins full and tortuous; nasal halves of both optic disks and temporal margins blurred by edema—though no measurable swelling ascertained. Lumbar puncture—bloody cerebrospinal fluid under increased pressure (approximately 19 mm.).

Treatment.—The presence of marked signs of high intracranial pressure associated with a perforating bullet injury of the brain and not associated with severe shock, made advisable a right subtemporal decompression and drainage, both for the purpose of lowering the increased intracranial pressure and thereby to prevent, if possible, a severe degree of medullary compression, but also to lessen the great danger of an infective meningitis and meningo-encephalitis resulting from the passage of the bullet with foreign material intracranially. The wound at the vertex was cleaned and packed with sterile gauze, so that the bleeding ceased; the longitudinal sinus had not been injured; brain tissue was oozing through the dural wound under high tension. An X-ray picture was taken while waiting for the operating room to be prepared (Fig. 112).

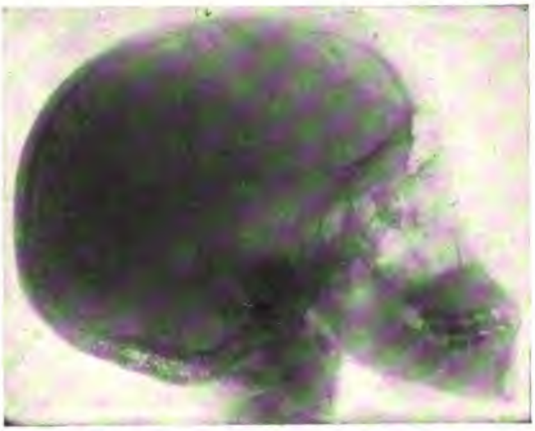


FIG. 112.—Lateral röntgenogram showing the bullet wound of entrance at the vertex and its course downward to its lodgment, just posterior to the foramen magnum; the right ventricle was perforated in its descent.

Operation (1 hour after admission and 4 hours after injury).—Bilateral decompression (no anesthesia being necessary): First, right subtemporal decompression; usual vertical incision, bone removed and no complications. Dura very tense and bluish and, upon incising it, bloody cerebrospinal fluid spurted to a height of almost 6 inches; upon enlarging the dural opening, the underlying cortex tended to protrude under high intracerebral pressure, causing the cortex to rupture in 3 places; multiple punctate hemorrhages throughout the cortex giving it the appearance almost of liver tissue. The cerebral pressure remained so high that the cortex protruded through the dural opening, so that a left subtemporal decompression was considered advisable to lessen this extreme intradural pressure. Usual closure with 3 drains of rubber tissue inserted. Temporary sterile gauze dressing applied.

Second.—Left subtemporal decompression: usual vertical incision, bone removed and no complications. The underlying dura was also very bluish and tense, and upon incising it, a supracortical hemorrhagic clot of 2 cm. in thickness was evacuated, permitting the underlying compressed cortex to rise. Numerous punctate hemorrhages throughout the cortex but no lacerations visible. Before the end of the operation, owing to the escape of much free blood and cerebrospinal fluid, the cortex pulsated feebly but still under tension. Usual closure with 2 drains of rubber tissue inserted. Duration, 85 minutes.

Post-operative Notes.—After the dura had been incised in the left subtemporal decompression, the patient opened his eyes and appeared to be conscious, although no anesthetic was necessary; again immediately after the operation, the patient appeared to be conscious and continued so for four minutes, when he became unconscious, and remained so. Four hours after operation, the temperature suddenly increased to 104.6°, the pulse to 130 and the respiration to 34, while the blood-pressure descended to 122; these signs of acute medullary edema rapidly progressed until patient died 12 hours after operation—a death typical of acute medullary edema.

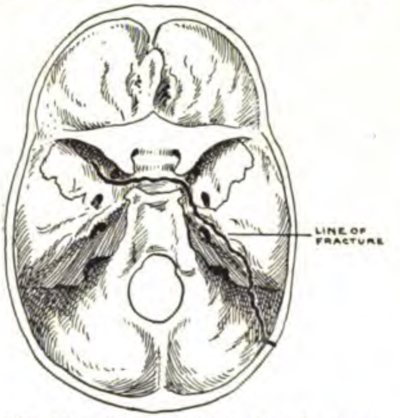


Fig. 113.—An extensive basilar fracture extending from the right occipital bone through the right petrous bone and across the sella turcica into the left middle fossa, in a patient dying from an acute medullary edema following a perforating bullet injury of the brain.

Autopsy.—The perforating bullet wound passed downward through the right ventricle and obliquely forward to lodge just posterior to the foramen magnum; the other bullet had passed transversely across the vertex of the vault but had not penetrated the dura nor injured the underlying longitudinal sinus. A line of fracture extended downward from the right mastoid bone obliquely forward along the petrous portion of the right temporal bone, across the sella turcica to a point just posterior to the left foramen rotundum (Fig. 113). The right lateral sinus had been torn by the fracture. No lines of fracture radiated from the wound of exit which was almost twice the size of the wounds of entrance. No extradural hemorrhage but a layer of subdural and supracortical hemorrhage of almost one-quarter of an inch in thickness—more over the left hemisphere. Extensive hemorrhagic clot in both ventricles. Much macerated brain tissue throughout the cerebral course of the bullet. A large amount of clotted blood beneath the tentorium as the result of the rupture of the right lateral sinus; the medulla itself was very edematous and "water-logged," being "jammed" downward into the foramen magnum.

Remarks.—In such an extensive and severe brain injury as occurred in this patient, the prognosis, both as to recovery of life and normality, is bad indeed; the only chance of recovery that this patient had lay in hope of a sufficient decompression and drainage to lessen the extreme i cranial pressure, and thereby prevent the onset of an acute medu edema. The autopsy findings, however, especially the rupture of the lateral sinus and the perforation of the right ventricle with profuse h rhage into both ventricles, made the recovery of this patient impos

All penetrating gunshot injuries of the skull, where the dura has been entered, should be treated as brain injuries associated with a fracture of the skull and with signs of increased intracranial pressure, as all of these patients have both an acute cerebral edema with more or less intradural hemorrhage; therefore, the treatment should be directed toward a lessening of this increased intracranial pressure by means of a subtemporal decompression and drainage, and if necessary a bilateral decompression and drainage; naturally the shock must be survived first to permit any operative procedure.

It was very interesting in this patient to observe the return to consciousness following the opening of the dura in the left subtemporal decompression, and thereby indicating a marked lessening of the high intracranial pressure; this relief was only temporary owing to the continued bleeding into the ventricles and subtentorially about the medulla. It is extremely rare for patients to survive longer than 12 hours following a hemorrhage into the ventricle. The slight impairment of the reflexes of this patient upon his admission to the hospital is very surprising and it merely emphasizes the necessity of considering the entire clinical picture rather than individual signs.

The line of fracture at the base passing through the right lateral sinus, the right petrous bone and across the sella turcica following a gunshot injury of the vertex with no lines of fracture extending downward from the vertex, indicates that this basal fracture was the result either of the explosive effect of the bullet injury (which is very doubtful and especially of this type) or that the patient in falling had struck his head violently against the floor or other solid object and the basal fracture resulted from it; this is the more probable explanation. If this complication had not occurred, the chances of the patient for recovery would have been greatly improved, although the ventricular hemorrhage is almost always a fatal complication.

Acute Severe Brain Injuries, with and Without a Fracture of the Skull, and Complicated by Meningitis. Death; Autopsy.

The complication of a purulent meningitis and meningo-encephalitis in patients having brain injuries usually results from an infection extending through the line of fracture by means of the nose, naso-pharynx or the ears in basal fractures, and in fractures of the vault in the presence of an overlying infected laceration or even contusion of the scalp, and especially if the adjacent dura has been torn; infected lacerations and hematomata of the scalp, even in the absence of an underlying fracture of the vault, may be a sufficient source of infection to extend intracranially by means of the diploetic veins and thus a purulent meningitis eventually appears; this complication occurs only too frequently following a cellulitis of the scalp—a most dangerous complication of cranial injuries.

In those patients having brain injuries associated with a high intracranial pressure sufficient to necessitate the operation of decompression and drainage, the complication of meningitis may not appear until five or six days following the decompression, and it is then always a question whether he meningitis has resulted from an inexcusable error in the operative hnic or from improper treatment of the tissues adjacent to the line of fracture into the nose, ears or the overlying scalp. The complication of meningitis, however, rarely occurs in these latter patients following early decompression and this is undoubtedly due to the early lessening of the increased intracranial pressure, so that these patients not only are less comatose and their general condition more resistant to infection, but the local tissues themselves have a more normal resistance to an infective process on account of their more normal circulation, whereas in stuporous and even unconscious patients without operation their general condition of resistance is quickly lowered and the local tissues become congested, boggy and edematous from the venous stasis resulting from the increased intracranial pressure and therefore less resistant to an infective process; besides, tissues under pressure are themselves not so capable of resisting an infective process as they are under the normal conditions of tension and circulation. This explanation may thus account for the greater freedom from an infective meningitis of patients whose increased intracranial pressure has been lowered early by the operation of decompression and drainage.

If, however, the early signs of an infective meningitis, and especially of the localized type, appear in a patient upon whom an operation has been performed or not, then an immediate subtemporal decompression and drainage should be advised in the hope that an early lessening of the increased intracranial pressure and the associated drainage would be sufficient to afford the patient a possible chance of recovery. No patient of this type, however, should be operated upon in whom the cerebrospinal fluid at lumbar puncture contains the bacterial organism itself, since this positive finding indicates the diffuseness of the infective process and the condition is therefore practically hopeless. If the signs of meningitis are recognized early, then the cerebrospinal fluid at lumbar puncture is frequently clear or only slightly turbid with an increased cell count due to the meningeal irritation and no bacteria can be found—these are the patients upon whom the operation of subtemporal decompression and drainage and even a suboccipital decompression and drainage combined with a laminectomy and drainage may offer a chance of recovery; it is only a chance, but it is worth taking as the risk is negligible compared with the condition itself, and although it is rare for the patient to recover, yet there are cases reported. I have only two patients that recovered following the operation of decompression and drainage out of a series of 14 who had all the signs of a purulent meningitis confirmed either at operation or at autopsy; neither of these two patients, however, exhibited the bacterial organism in the cerebrospinal fluid at lumbar puncture.

Acute severe brain injuries associated with a fracture of the skull; meningitis. Death; autopsy.

A. *No marked signs of intracranial pressure and therefore no operation having been performed.*

Case 80.—Acute severe brain injury associated with a subdural hem rhage and a fracture of the skull, but no signs of high-intracranial press No operation. Meningitis; death. Autopsy.

No. 145.—Agnes. Thirty-one years. White. Married. Housework.

Admitted May 28, 1914, Polyclinic Hospital. Referred by Doctor John A. Bodine.

Died June 4, 1914—6 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While hanging out clothes from a fire-escape, patient fell a distance of 12 feet upon the stone pavement below; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (40 minutes after injury).—Temperature, 97.6°; pulse, 82; respiration, 20; blood-pressure, 134. Semiconsciousness and in shock. Extensive laceration over left occipital area down to bone; careful probing did not reveal an underlying fracture. No bleeding from nose. mouth or ears; no mastoid ecchymoses. Pupils equal and react normally. Reflexes slightly depressed but otherwise negative. Fundi negative.

Treatment.—Expectant palliative; laceration of scalp widely shaved, cleaned and loosely sutured with one drain inserted. Careful observation instituted, but it was not considered that the patient was seriously injured intracranially. Within a few hours, the signs of shock disappeared and the general condition of the patient improved so that an excellent prognosis was given. Twenty-four hours after admission, patient complained of some headache and backache and an impairment of the left ear; otoscopic examination revealed a bluish discoloration of the left tympanic membrane, and it was then for the first time that a fracture of the skull was suspected, and there were present no signs of an increased intracranial pressure. (The lesion of the left middle ear was demonstrated by the sound of the tuning fork being always referred to the left ear—Weber's test, and the bone conduction of the left ear being always greater than air conduction—Rinne's test.) With the exception of impairment of hearing of the left ear, patient seemed to be making an excellent recovery until June 3 (5 days after injury), when the patient complained of severe headache, was nauseated for several hours and finally vomited; the temperature was 100.2°, pulse 80, respiration 20, blood-pressure 136; the physical examination was negative except that the ophthalmoscope revealed enlarged retinal veins but no blurring of the details of either optic disk. This condition continued until 18 hours later, when the patient could not be aroused by the nurse and the following examination was made:

Examination (6 days after injury).—Temperature, 103.8°; pulse, 120; respiration, 32; blood-pressure, 140. Profound unconsciousness. Slight stiffness of the neck but no Kernig reflex. No ocular paralyses nor convulsions. Pupils slightly enlarged but equal and react to light sluggishly. Reflexes—patellar active but equal; no ankle clonus nor Babinski; abdominal reflexes cannot be elicited. Fundi—retinal veins enlarged; no blurring of the optic disks. At this stage of the examination, the respiration suddenly became irregular and then ceased; artificial respiration and pulmotor continued life for 4 hours—pulse rising to 160 plus and finally became imperceptible; patient died 6 days after injury.

Autopsy (Doctor O. H. Schultze).—A line of fracture of the bursting type began in left occipital bone about 2 cm. below the lacerated area of the scalp and extended downward and forward into and along the crest of the left petrous bone to the sella turcica where it stopped; another line of fracture extended through the left orbital plate and the left greater wing of the sphenoid bone—either fracture line being possibly the source of the infection (Fig. 114). Subperiosteal hemorrhages were situated over both orbital plates. Covering the entire left hemisphere and the upper portion of the right hemisphere was a thin creamy purulent exudate (bacteriological report (Doctor Jeffries)—"staphylococcus"); much thick creamy pus in the base, especially in the middle and posterior fossæ. Cortical laceration and thin supracortical hemorrhage over right frontal lobe and tip of right temporosphenoidal lobe. Small cortical lacerations over the posterior portion of left cerebellar lobe—underlying the point of contact. Ventricles negative.

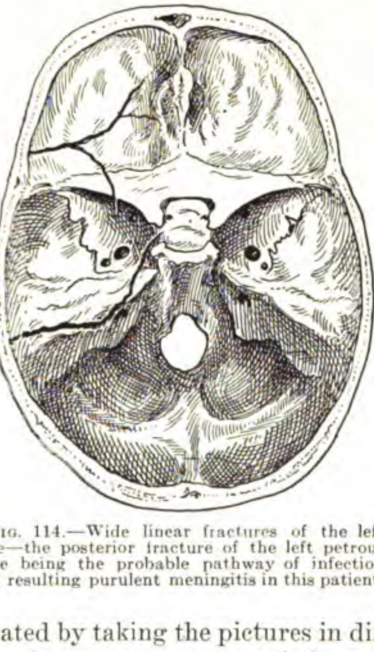


FIG. 114.—Wide linear fractures of the left base—the posterior fracture of the left petrous bone being the probable pathway of infection and resulting purulent meningitis in this patient.

Remarks.—This is a most instructive case: if a meningitis had not occurred and the patient had recovered (as was our belief on the third day after admission), the diagnosis would have been a "possible fracture of the skull" or even a "severe concussion with laceration of the scalp." No doubt many brain injuries, with or without fractures of the skull, are thus diagnosed, and it is only when complications occur necessitating an operation, or if death should occur and an autopsy is performed, then the true condition is ascertained. A röntgenogram should have been taken of this patient, and it is possible that the occipital fracture would have been located by taking the pictures in different planes and angles; this would have been important not so much from the standpoint of treatment as to whether a cranial operation should be performed or not, but to warn the medical attendant that the cranial injury may be of greater severity than a mere "bump" upon the head and the so-called "concussion"—a much overworked term and applied much too frequently. Then again, a lumbar puncture should have been performed as soon as the patient had recovered from the signs of shock; if blood had been found in the cerebrospinal fluid, even in the absence of an increased intracranial pressure, it would have at least informed us that the intracranial condition was one of greater moment than a mere "concussion." Besides, a lumbar puncture should have been pe
formed surely on the fifth day after admission, when the patient compla
of severe headaches and even vomited, and especially when the tempe
had risen from 99° to 100.2°; the fact that the ophthalmoscopic e

tion was practically negative should not have lulled us into the feeling that the condition was possibly of intestinal origin. From the standpoint of not having had an X-ray picture taken, and especially the neglect to have a lumbar puncture performed—these two oversights and mistakes are strongly reprehensible and should not be permitted to occur.

The source of the purulent meningitis was undoubtedly through the left middle ear (even though the left tympanic membrane was intact), although the laceration of the scalp being in close proximity to the fracture of the occipital bone might have been a possible source of the infection; the laceration of the scalp, however, was "clean," and the dura underlying the occipital fracture was not involved by the meningitis.

The autopsy findings of cortical lacerations of the anterior surface of the right frontal lobe and of the right temporo-sphenoidal lobe would indicate cerebral *trauma au contre-coup,* whereas the small laceration of the posterior portion of the left cerebellar lobe immediately underlying the site of the cranial injury—the area of contact, would be the result of the direct local injury. These cortical lacerations of comparatively silent areas of the brain are relatively unimportant, unless the resulting hemorrhage and cerebral edema are of such large amount that the intracranial pressure is greatly increased and therefore necessitating the operation of cranial decompression and drainage. These latent cortical lesions are undoubtedly of much greater frequency than is usually realized, and it is only by careful autopsy examinations that many of them are even suspected.

It would seem from the autopsy findings that the meningitis in this patient had resulted from an extension of infection through the left middle ear into the subdural spaces; the onset was so slow and insidious that its presence was not suspected until the process had become so diffused throughout the cerebrospinal canal that there was practically no chance for the patient to recover by any known method of treatment. At the time, the advisability of performing an unilateral, and better, a bilateral decompression and drainage, and even a high laminectomy with drainage was considered, but the general condition of the patient became so weakened that even this possible means of treatment was not used; no matter what the treatment, operative or not, the end-result is the same in practically all of these patients—once bacteria are found in the cerebrospinal fluid at lumbar puncture following a cranial meningitis.

This patient illustrates the advisability of repeated lumbar punctures if the condition is not improving as rapidly as should be expected; especially is this so in the presence of a fracture which opens into the ears, nose or pharyngeal cavities, and particularly if the fracture underlies a laceration of the scalp; the danger of the formation of single or multiple cerebral abscesses in these patients developing a localized meningitis is very great indeed. The absence of a purulent discharge from the left ear as a forerunner of the infective process extending inward through the line of fracture is rather unusual; it is possibly unfortunate that a blockage occurred at the intact left tympanic membrane in this patient—thus facilitating the extension of the infection inward by not permitting free drainage. It usual that there were no local irritative signs of the left cortical involvement—usually recognized by convulsive seizures; greater reliance, however, should be placed upon the cell count of repeated daily lumbar punctures—the most accurate method of anticipating meningeal involvement.

B. *Signs of high intracranial pressure and therefore the operation of cranial decompression having been performed.*

CASE 81.—Acute severe brain injury associated with signs of high intracranial pressure due to subdural hemorrhage; fracture of the left vault and base. Left subtemporal decompression and drainage. Meningitis. Death. Autopsy.

No. 103.—Joseph. Fifty years. Colored. Married. Coachman. U. S.

Admitted January 18, 1914, Polyclinic Hospital. Referred by Doctor J. P. Grant.

Operation (January 18, 1914—6 hours after injury).—Left subtemporal decompression and drainage.

Died February 11, 1914—23 days after injury and operation.

Family history negative.

Personal history negative.

Present Illness.—While driving his carriage, patient collided with a pillar of the elevated railroad and was hurled headlong against the stone curbing; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (40 minutes after injury).—Temperature, 99.2°; pulse, 62; respiration, 16; blood-pressure, 168. Unconscious, but can be aroused with difficulty; no alcoholism. Abrasion of left side of face, which is very much swollen and ecchymosed. No bleeding from nose, mouth or ears. Pupils moderately enlarged but react normally. Reflexes—patellar exaggerated—right more than left; no ankle clonus but right Babinski; abdominal reflexes absent. Fundi—retinal veins enlarged; nasal margins of both optic disks blurred, left possibly more than right.

Treatment.—Expectant palliative. In spite of this treatment, the signs of an increasing intracranial pressure became more marked—the pulse descended to 54 and the respiration to 14 and of the irregular Cheyne-Stokes character, while the blood-pressure increased to 170; one hour after admission, patient had typical "projectile" vomiting—without nausea, he would open his mouth and strike the wall, a distance of three feet away; his unconsciousness became more profound until the following examination was made:

Examination (4 hours after admission).—Temperature, 100.8°; pulse, 52; respiration, 14; blood-pressure, 170. Profound unconsciousness and cannot be aroused. Large hematoma over left side of face; no fracture of the jaw can be ascertained. Pupils—left larger than right and reacts to light sluggishly. Reflexes—patellar markedly exaggerated, right very much greater than left with right patellar clonus; right ankle clonus and right Babinski; right abdominal reflex absent. Fundi—retinal veins dilated; entire left optic disk blurred by edema but no measurable swelling, while nasal half and temporal margin of right optic disk obscured. Lumbar puncture—bloody cerebrospinal fluid under high pressure (approximately 20 mm.). The spasticity of right arm and right leg had now developed into a definite weakness of the entire right side of the body (as well as could be elicited in a patient so profoundly unconscious).

Treatment.—A left subtemporal decompression was immediately advised to prevent a greater medullary compression and thereby avoid the danger of the onset of an acute medullary edema.

Operation (5 hours after admission).—Left subtemporal decompression (no anesthesia being necessary): usual vertical incision, bone removed and no complications; bone itself unusually thick and like ivory. Dura under high tension, and upon incising it, blood-tinged cerebrospinal fluid spurted a distance of 12 inches; upon enlarging the dural opening, much bloody cerebrospinal fluid escaped, exposing a very "wet" edematous cortex which soon pulsated normally. No cortical hemorrhages or lacerations visible. Usual closure with 2 drains of rubber tissue inserted. Duration, 50 minutes.

Post-operative Notes.—Patient made an excellent operative recovery—becoming conscious 30 hours after operation, and although mildly irrational for 2 days, he seemed on the road to an excellent recovery; all sutures were removed on the sixth day post-operative. Eight days after operation, patient developed a temperature of 104°, and then the signs of an infection of the hematoma over the left side of the face appeared in that it became unusually tender, painful and feverish; no fluctuation obtained, however; it was incised and carefully treated by Doctor John P. Grant and by Doctor J. A. Robertson—the culture showing pure growth of a short-chained streptococcus; in spite of this vigorous treatment, the patient developed the signs of meningeal irritation—the operative decompression area bulged, temperature ascended to 107° and the patient eventually died from the signs of a typical meningitis on the twenty-third day after injury and operation; a lumbar puncture had obtained a cloudy cerebrospinal fluid—a "pure growth of a short-chained streptococcus."

Autopsy.—The 2 incisions of the hematoma over left side of face did not contain any frank pus. Decompression wound healed perfectly. Fracture of skull extending from the anterior margin of decompression opening downward into left anterior fossa of the skull—a distance of 4 inches (Fig. 115). No extradural hemorrhage, but a thin layer of subarachnoid hemorrhage over entire left frontal lobe extending backward to the left Rolandic fissure. Thick plastic exudate over the entire left temporo-sphenoidal lobe; very thick creamy-yellow pus with foul odor at the base of operative field and in the middle fossa, where there were almost 6 ounces of it. The infection was limited to left side of brain—the right hemisphere being negative. A probe could be passed from temporal muscle of operative field downward into infected area of hematoma beneath left zygomatic arch—a very possible means of the extension of the infection of the hematoma. (Cultures of this intracranial pus showed both streptococci and staphylococci.) Ventricles negative.

Remarks.—In the presence of skull fractures in close proximity to extensive hematomata, either of the scalp or of the adjacent tissues of the face and neck, it is always wiser and safer to incise the hematoma early under aseptic conditions, so that future infection of the hematoma is lessened and the great danger of an infective extension from the hematoma through the fracture of the skull intracranially is thus avoided. This precaution is particularly advisable in cases of hematoma of the scalp having a fracture of the underlying bone; large hematomata are particularly susceptible to infections through the skin, especially when they are under tension, and if they do become infected, then the danger of this infective process extending through the underlying fracture is very great indeed. Undoubtedly this patient would have recovered if this complication could have been anticipated; at no time, however, was there any fluctuation of the hematoma to be obtained—merely a boggy edema of the tissues and therefore no incision had been considered advisable. During the past 2 years, all hematomata of the scalp were incised early under aseptic precautions whenever there was an adjacent fracture of the skull, and during this period of 2 years a meningitis complicating the recovery has not yet occurred. The autopsy findings demonstrate the cause of the weakness of the right side of the body in that a layer of subarachnoid hemorrhage was found over the entire left cortical hemisphere anterior to the fissure of Rolando. Why convulsions of the localizing type did not occur cannot be explained, unless the general intracranial pressure was so high as to inhibit the irritative effect of the cortical lesion.

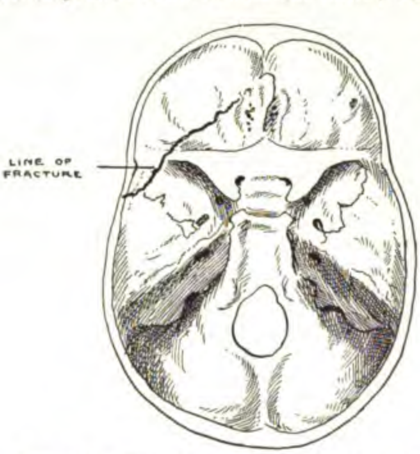


Fig. 115.—Wide linear fracture of left anterior fossa of the base of the skull—a very probable source of infection and resulting meningitis in this patient—either from the infected hematoma of the left side of face or through the cribriform plate of the ethmoid bone.

It is again unfortunate in this patient that a lumbar puncture was not performed upon his admission to the hospital as it is possible that a definite increase of the intracranial pressure would have been ascertained then and an earlier operative relief would have been afforded; the absence of shock would have made this a safe procedure. However, with the later operation the patient made an excellent recovery, but that does not always occur following these delayed operations, for it is then frequently too late for the patient to recover.

Case 82.—Acute severe brain injury associated with high intracranial pressure due to subdural hemorrhage and cerebral edema; fracture of the vault. Left subtemporal decompression and drainage. Meningitis. Death. Autopsy.

No. 115.—Frederic. Thirteen years. White. School. U. S.

Admitted January 28, 1914, Polyclinic Hospital. Referred by Doctor A. S. Morrow.

Operation (January 30, 1914—40 hours after injury).—Left subtemporal decompression and drainage.

Died February 9, 1914—10 days after operation and 12 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While playing in a school yard, the patient fell a distance of nine feet, striking his head upon the stone pavement; no immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (1 hour after injury).—Temperature, 97.6°; pulse, 96; respiration, 28; blood-pressure, 108. Semiconscious and in severe shock. Large hematoma over left side of head; definite fluctuation. No bleeding from nose, mouth or ears. Pupils slightly enlarged but react normally. Reflexes—patellar active but equal; no ankle clonus but right Babinski; abdominal reflexes absent. Fundi negative.

Treatment.—Expectant palliative; vigorous shock measures instituted. Within 30 minutes, although the pulse had ascended to 120, the temperature had increased to 99° and the blood-pressure to 120 so that it could be said that the general condition had improved. Patient remained in a semiconscious condition for 36 hours, when the signs of an increasing intracranial pressure appeared.

Examination (37 hours after admission).—Temperature, 100.6°; pulse, 80; respiration, 18; blood-pressure, 118. Patient has become more stuporous and when aroused is irrational; holds hands to head as though in pain. Hematoma over the left side of head very tense. Pupils—left larger than right and does not react to light. Reflexes: patellar—right more active than left; right exhaustible ankle clonus and right Babinski; abdominal reflexes present, right possibly less active than left. Fundi—retinal veins enlarged; nasal halves of both optic disks obscured by edema—temporal margins, however, clear. Lumbar puncture—blood-tinged cerebrospinal fluid under high pressure (approximately 17 mm.). X-ray report (Doctor A. J. Quimby)—"two lines of fracture extending irregularly through squamous portion of left temporal bone."

Treatment.—An immediate left subtemporal decompression advised to lessen the increasing intracranial pressure.

Operation (39 hours after admission).—Left subtemporal decompression; usual vertical incision, bone removed and no complications; much free blood and several clots in the temporal muscle itself beneath the temporal fascia and therefore a fracture of the underlying bone was to be expected; 2 irregular lines of fracture extended obliquely through the underlying squamous bone; much free blood escaped through the lines of fracture, though no definite extradural hemorrhage was ascertained upon removing the bone. Dura very tense and upon incising it, blood-tinged cerebrospinal fluid spurted a distance of 2 inches for a period of 5 seconds; upon enlarging the dural opening, much bloody cerebrospinal fluid escaped, allowing the underlying bulging cortex to recede and to pulsate normally at the end of the operation. No cortical hemorrhage or laceration visible—merely a very "wet" edematous brain. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Patient made an excellent operative recovery; within 12 hours, the pulse ascended to 80 and remained at this level; complete return of consciousness and the patient merely complained of a dull throbbing headache. At the first dressing—44 hours after operation—one drop of pus (staphylococcus upon culture) was found. On February 5—6 days after operation—the temperature suddenly ascended to 103°, and upon removing the sutures, a small amount of cerebrospinal fluid escaped mingled with pus (staphylococcus); pulse ascended to 138, neck became rigid and a positive Kernig reflex was elicited; the cerebrospinal fluid at lumbar puncture was cloudy (staphylococcus also upon culture). Patient now became progressively worse and died from a diffuse meningitis—10 days after operation and 12 days after injury.

Autopsy.—Two small lines of fracture extended from the anterior edge of left decompression opening forward and downward into the anterior and middle fossæ of the base of the skull (Fig. 116). Thick creamy pus (staphylococcus upon culture) covered not only the left hemisphere but also the right hemisphere and extended into the subtentorial fossa; the decompression wound was thoroughly saturated with this pus. Several small pieces of bone wax which had been used to control the bleeding from the edges of the bony opening were found in excess. Bacteriological examination of the bone wax used in the operating-room was now made and showed the presence of staphylococci, and thus indicating the mode of infection. (Through a mistake, this bone wax had been considered sterile, so that it had not been boiled before the operation and therefore it was undoubtedly the cause of infection and death of this patient; this inexcusable error should not have been permitted to occur and it is one of those unfortunate mistakes occurring only too frequently in a large hospital but it is not likely to be repeated.)

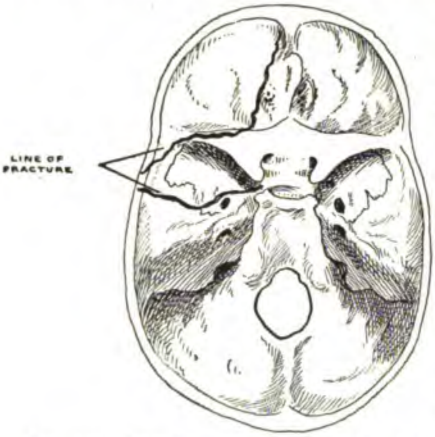


FIG. 116.—Two linear fractures of the left middle and anterior fossæ in a patient dying from a purulent meningitis—an infection resulting from a lack of surgical asepsis in the use of non-sterile bone wax.

Remarks.—If no cause for the infecton in this case had been ascertained at autopsy, such as the bone wax, the source of infection would have undoubtedly been considered as due to an infection of the hematoma of the scalp overlying the lines of fracture; in this patient, however, the hematoma seemed to be subpericranial and therefore the danger of its becoming infected from the scalp was not so much to be feared—unless the overlying tissues were severely contused or lacerated.

It is interesting in this youthful patient of 13 years of age to note that the high intracranial pressure as revealed by the ophthalmoscope and the lumbar puncture could not cause clinically the marked signs of a similar high intracranial pressure in adults, and this clinical syndrome is characteristic in children in whom it is rare to have a pulse-rate lowered below 60 and a respiration-rate below 16, even in the presence of severe intracranial pressure and medullary compression of high degree. Children are thus enabled to withstand the effects of high intracranial pressure much better than adults, and for this reason the operation of cranial decompression is less urgent in the majority of these youthful patients, because the onset of an acute medullary edema is not so much to be feared; besides, their general absorptive ability "to take care of" an increased intracranial pressure due to subdural blood and cerebral edema is much greater than that of adults. The condition of shock, however, is apparently of greater significance in children than in adults, so that the most vigorous shock measures should be instituted as early as possible and thus enhance their chances of recovery.

The importance of having an autopsy performed upon this patient cannot be overestimated in that the cause of death was accurately ascertained as being due to a careless oversight—one of ignorance, and thus future patients were spared this unnecessary risk. The cause of death should be ascertained by autopsy of each patient who dies, and it is only fair to future patients that these causes, if avoidable, should be naturally eliminated. Permission in writing for autopsy should be obtained before operation in each patient—from the nearest relative, and in this manner "mistakes" should not occur more than once.

A recovery of life of this patient might have been obtained if, as soon as the appearance of a local infection of the wound occurred, a small drainage incision had been made, and if necessary, the entire wound reopened and excellent drainage afforded—similar to the treatment of an early localized meningitis following otitic disease for which an ipsolateral decompression and drainage is performed and with excellent results in the early cases; during the past year this has been possible in several patients.

Case 83.—Acute severe brain injury associated with signs of high intracranial pressure due to subdural hemorrhage and cerebral edema. Right subtemporal decompression and drainage. Meningitis; left subtemporal decompression. Death. Autopsy.

No. 671.—Charles. Thirty-five years. White. Married. Porter. Scotland. Admitted September 1, 1916, Polyclinic Hospital.

Operations, 1st (September 1, 1916—8 hours after injury).—Right subtemporal decompression and drainage. 2nd (September 20, 1916—19 days after first operation).—Left subtemporal decompression and drainage.

Died September 22, 1916—21 days after admission and 2 days after the second operation.

Family history negative.

Personal history negative.

Present Illness.—While standing upon some stone steps, the patient slipped and fell a distance of 7 feet, striking his head against the stone pavement; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (45 minutes after injury).—Temperature, 98°; pulse, 120; respiration, 28; blood-pressure, 122. Unconscious and in severe shock. Contusion of scalp over the occipital protuberance and vertex of skull. No bleeding from nose, mouth or ears; no mastoid ecchymoses. Pupils equally dilated and do not react to light. Reflexes all absent, except slight corneal reflex. Fundi negative.

Treatment.—Expectant palliative; vigorous shock measures instituted. Within 2 hours after admission, the general condition of the patient markedly improved—the temperature rose to 99.4°, while the pulse decreased to 82 and the respiration to 18, and the blood-pressure ascended to 138; 3 hours after admission, both plantar reflexes returned. Within the next 3 hours, however, the signs of an increasing intracranial pressure appeared more and more marked.

Examination (6 hours after admission).—Temperature, 99.8°; pulse, 48; respiration, 14; blood-pressure, 140. Semiconscious and restless. Bogginess over right temporal area with slight right mastoid ecchymosis. Pupils equal and react normally. Reflexes—patellar active but equal; slight double exhaustible ankle clonus and a double suggestive Babinski; abdominal reflexes present and equal. Fundi—retinal veins enlarged; nasal halves of both optic disks blurred by edema. Lumbar puncture—bloody cerebrospinal fluid under high pressure (approximately 19 mm.).

Treatment.—Owing to the signs of high intracranial pressure, as indicated by the marked lowering of the pulse- and respiration-rates and by the ophthalmoscopic and lumbar puncture examinations, a right subtemporal decompression was advised—the patient being right-handed and there being no localizing signs of a greater intracranial pressure over one hemisphere than the other.

First Operation (7 hours after admission).—Right subtemporal decompression: usual vertical incision, bone removed and no complications; much free blood in temporal muscle beneath the temporal fascia and therefore upon retracting the muscle, a fracture of the underlying squamous bone was exposed—slightly depressed. Large extradural hemorrhage evacuated, revealing a tense underlying dura, especially in the upper portion of the field. Upon incising the dura, bloody cerebrospinal fluid spurted a distance of 3 inches, revealing a very "wet," edematous cortex with one laceration of about 2 cm. in length in the upper temporal convolution. Small layer of subarachnoid and possibly cortical hemorrhage had occurred over the middle temporal convolution. Much free subdural blood and cerebrospinal fluid escaped during the operation, permitting the protruding cortex to recede and to pulsate at the end of the operation. Usual closure with 2 drains of rubber tissue inserted. Duration, 50 minutes.

Post-operative Notes.—Patient made an excellent operative recovery in that he became conscious and rational within 2 days, pulse ascended to 66 and the respiration to 18, while the signs of an increased intracranial pressure became less marked. Decompression area continued to bulge tensely and the patient complained of persistent daily headaches. Nineteen days after operation, patient had a convulsion beginning in the right arm and extending to the right leg and face and then merged into a general convulsion; the following examination was now made:

Examination (19 days after admission and operation).—Temperature, 103.2°; pulse, 96; respiration, 26; blood-pressure, 148. Decompression wound bulging and does not pulsate. Pupils both contracted and do n react to light. Reflexes—patellar very much exaggerated, right possibl more than left; no ankle clonus nor Babinski; abdominal reflexes could n be obtained. Fundi—retinal veins dilated; double papilledema of one diopter swelling—entire retinæ being congested and suffused. Lumbar puncture—slightly turbid cerebrospinal fluid under high pressure (approximately 17 mm.); bacteriological examination (Doctor Jeffries)—"large number of leucocytes but no bacteria."

Treatment.—In the presence of a high intracranial pressure and a large number of leucocytes but no bacteria in the cerebrospinal fluid, it was considered advisable to perform a left subtemporal decompression and drainage in the hope that the meningitis might remain localized and thus the patient be given a chance of recovery.

Second Operation (19 days after first operation and injury).—Left subtemporal decompression and drainage: usual vertical incision, bone removed and no complications; about one inch above the posterior base of the left zygomatic process was a bony enlargement of the squamous bone and upon rongeuring into it a cavity filled with a cheesy deposit was removed (possibly an isolated zygomatic cell of the mastoid and of the size almost of a lima bean). Dura very tense and upon incising it, slightly straw-colored cerebrospinal fluid spurted a distance of 3 inches, and upon enlarging the dural opening a very "wet," edematous cortex was exposed. In the sulci about the vessels was a whitish exudate beneath the arachnoid but no free pus observed. Cortex tended to protrude but did not rupture, owing to the rapid escape of a large amount of cerebrospinal fluid. Usual closure with 2 drains of rubber tissue inserted. It was decided to explore the first decompression incision and upon doing so much free pus was found beneath the temporal fascia among the fibres of the temporal muscle; 2 drains of rubber tissue inserted. (Bacteriological report (Doctor Jeffries)—"staphylococci.") Duration, 1 hour.

Post-operative Notes.—Much yellowish pus drained from the first operative incision and after 12 hours pus appeared in the drainage from the second decompression. Patient's condition rapidly became worse—rigidity of the neck occurring within 12 hours after operation, simultaneous with the appearance of a double Kernig reflex, while the temperature ascended to 107° and the patient died—40 hours after the second operation and 21 days after injury.

Autopsy.—A linear fracture extended downward from the right decompression opening into the middle fossa, but did not reach the sella turcica (Fig. 117). Diffuse subdural meningitis and a layer of creamy pus was found over the entire brain—more over the right hemisphere, and also in the ventricles. No gross hemorrhage ascertained.

Remarks.—The infection causing the meningitis of this patient undoubtedly occurred at the time of the first operation and it is naturally inexcusable. It is interesting to note the long period of incubation necessary for a diffuse meningitis to occur, and I believe this is due to the fact that the increased intracranial pressure had been so lessened by the decompressed opening that the underlying tissues were therefore not under high pressure and thus their resistance to the infection was all the greater. It is unfortunate that the true condition of the localized meningitis beneath the site of the first operation could not have been ascertained earlier, so that adequate drainage could have been effected and thus the localized meningitis be satisfactorily drained with the ultimate recovery of the patient; the clinical signs, however, of a meningitis occurred so suddenly "out of a clear sky," as it were, that it was then too late for satisfactory drainage to be instituted.

The second operation of left subtemporal decompression and drainage was advised chiefly from the fact that the bacteriological report of the cerebrospinal fluid was negative for bacteria—only the presence of leucocytes being demonstrated; if bacteria had been found at lumbar puncture, then not even a decompression would have been attempted, for it would have been realized that the meningitis was therefore an extensive and a diffuse process and beyond satisfactory drainage by operation. Patients, however, having merely a meningeal irritation or a localized meningitis and whose cerebrospinal fluid at lumbar puncture does not contain bacteria (even though turbid and cloudy, which may be due to aggregations of leucocytes) —these patients should be given the chance of recovery by means of a unilateral and, if necessary, a bilateral subtemporal decompression and drainage; this is particularly true following brain injuries and the meningeal complications of middle ear and mastoid disease.

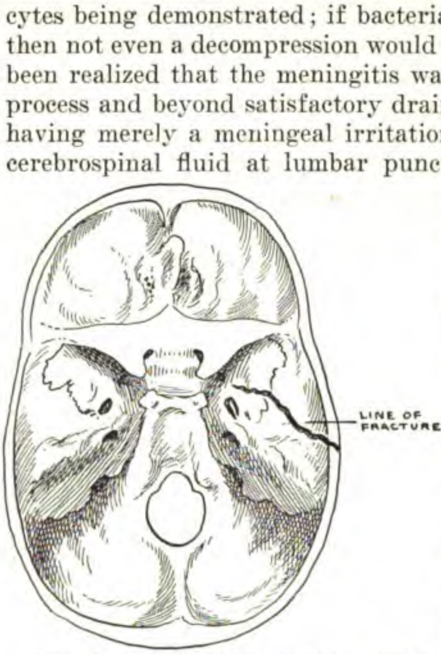


Fig. 117.—Wide linear fracture extending into right middle fossa in a patient dying from a purulent meningitis, resulting from the introduction of bacteria at the time of the right subtemporal decompression—inexcusable and most careless.

The marked contraction of both pupils and their non-reaction to light at the examination just before the second operation indicated the cortical irritation due to the meningeal exudate and the meningo-encephalitis; as this supracortical pressure increased, then the irritative effect would be submerged by the paralytic effect of high supracortical pressure, and thus the pupils would become dilated and likewise non-reacting to light. This latter stage of pupillary dilatation must not be confused with that dilatation which so frequently occurs immediately following the cranial injury—that is, the pupillary dilatation due to the severe shock of the injury; naturally, the other signs of shock are present, particularly the subnormal temperature and the lowered blood-pressure of shock.

C. *Subtemporal decompression and drainage performed after the signs of meningitis had appeared.*

Case 84.—Acute severe brain injury with no signs of high intracranial pressure; fracture of base of skull. Meningitis; right subtemporal decompression and drainage. Death. Post-mortem examination.

No. 032.—John. Twenty-four years. White. Single. Farmer. U S

Admitted April 10, 1913—30 days after injury, Muhlenburg Ho

Plainfield, N. J. Referred by Doctor Van Horn.

Operation (June 8, 1913—58 days after admission and 88 days after injury).—Right subtemporal decompression and drainage.

Died June 22, 1913—14 days after operation, 72 days after admission, and 102 days after injury.

Family history negative.

Personal history negative.

Present Illness.—Thirty days before admission, patient was struck over the head by a large wooden beam; immediate loss of consciousness; treated at home with the expectant palliative method. His general condition improved, although the bloody discharge from the right ear continued for almost one week and then it became a thin watery, purulent discharge; on account of being "feverish" and complaining of dull headache, associated with dizziness, patient was brought to the hospital for treatment.

Examination upon admission (30 days after injury).—Temperature, 99°; pulse, 72; respiration, 20; blood-pressure, 136. Rather drowsy but otherwise normal; no external signs of scalp injury. Thin purulent discharge from right ear; no mastoid ecchymosis or tenderness. Bacteriological report—numerous streptococci. Pupils equal and react normally. Reflexes—patellar exaggerated but equal; no Babinski. Fundi negative. X-ray—negative report.

Treatment.—Expectant palliative.

Examination (58 days after admission and 88 days after injury).—(Upon a visit to the hospital to operate upon another patient, I was asked to examine this patient whose condition had not improved but had become worse several hours ago.) Temperature, 99.4°; pulse, 88; respiration, 26; blood-pressure, 134. Confused mentally and complains of severe frontal and occipital headaches, and "I feel chilly all over." Definite rigidity of neck with double positive Kernig reflex. Pupils equal and react normally. Reflexes—patellar very much exaggerated—left possibly greater than right; exhaustible left ankle clonus and left Babinski; abdominal reflexes present and equal. Fundi—retinal veins dilated; papilledema of both optic disks having a measurable swelling of 1 diopter. Lumbar puncture—slightly turbid cerebrospinal fluid under increased pressure (approximately 16 mm.); bacteriological report—occasional streptococcus.

Treatment.—In the belief that an early right subtemporal decompression, exploration and drainage might still be sufficient to obtain a recovery of life, and in the hope that the condition of mild meningitis might be due to a brain abscess of the right temporo-sphenoidal lobe, a right subtemporal decompression was advised as the patient's only chance of recovery of life. (The presence of streptococci in the cerebrospinal fluid at lumbar puncture, we now know, made the prognosis practically hopeless as far as a recovery of life might be obtained by any operative means of drainage, etc.; and even if a recovery of life could be obtained under these conditions, the ultimate result would not be a normal individual.)

Operation (58 days after admission and 88 days after injury).—Right subtemporal decompression, exploration and drainage: usual vertical incision, bone removed and no complications. Dura very tense and upon incising it, turbid cerebrospinal fluid escaped under high pressure (bacteriological report—numerous streptococci); upon enlarging dural opening, a congested and slightly hazy cortex tended to protrude but did not rupture; in the sulci about the vessels was a distinct whitish subarachnoid exudate. A ventricle puncture needle was used to locate, if possible, a brain abscess, but all attempts were unsuccessful. So much cerebrospinal fluid had escaped during the operation that the cortex had become slightly relaxed, so that it pulsated. Usual closure with 2 drains of rubber tissue inserted. Duration, 50 minutes.

Post-operative Notes.—Patient drained freely through the operative incision, became conscious after 24 hours and at the end of 56 hours the temporal margins of both optic disks were visible at an ophthalmoscopic examination and the general condition of the patient so improved, during the following 6 days, that an ultimate recovery of life was even expected. On the eighth day post-operative, the temperature suddenly ascended to 106°, pulse to 138, and the respiration to 36; patient became comatose, and this condition steadily progressed until the patient died, 14 days after operation.

Post-mortem Examination.—An examination through the operative incision revealed a diffuse meningitis with much free pus in the middle fossa. No brain abscess could be found.

Remarks.—The condition of meningitis in the patient undoubtedly developed from the right middle ear infection—this complication occurring in a very small percentage of the cases, especially when the ear is irrigated, plugged or otherwise "meddled" with. The diagnosis of a possible brain abscess was due chiefly to the subacute character of the meningitis and also to the mild increased intracranial pressure to the extent of only a papilledema of one diopter of swelling; it is rare for the condition of brain abscess by itself to produce the condition of high intracranial pressure, and especially that of "choked disks" of more than 2 diopters of swelling, because brain abscesses replace brain tissue by substitution and therefore they do not tend to increase the intracranial pressure as do brain tumors (the gliomatous tumors excepted, which also replace brain tissue); if the brain abscess should cause a meningeal irritation to the extent of even a meningitis by direct extension or rupture into the ventricles so that a diffuse meningitis occurs, then the intracranial pressure may be increased to an extreme degree by either a blockage of the ventricles and thus producing an internal hydrocephalus, or by blocking the normal channels of excretion of the cerebrospinal fluid into the cortical veins, sinuses, etc., and thus in reality an external hydrocephalus results. As a point, therefore, in the differential diagnosis of brain abscess and brain tumor, the presence of "choked disks" of 2 diopters and more always tends to indicate the condition of brain tumor rather than brain abscess, even if the abscess is cerebellar and there are no marked signs of meningeal irritation—and especially in the absence of a meningitis.

It is unfortunate that a complete autopsy of the head could not h
been obtained in this patient; these examinations through cranial ope
incisions are most unsatisfactory and very little can be ascertained by
of them; if permission is obtained before the operation (and it alwa
be in these patients), then it would always be possible to prev
operative mistakes from occurring and it would also aff
to ascertain the true intracranial condition for th
of future patients.

A right subtemporal decompression and exploration was performed upon this patient chiefly because the line of fracture extended through the right ear from which a purulent discharge had persisted for several weeks, and also to the increased reflexes of the left side of the body—particularly of a left Babinski. In the presence of a purulent meningitis, the site of the fracture of the skull is of real importance and is an aid to the localization of the intracranial lesion.

It was a distinctly dangerous procedure, and even unsurgical, to have used a ventricle puncture needle in an effort to locate a subcortical cerebral abscess when it was necessary to pass this needle through the visible subarachnoid purulent exudate; if the abscess was not found, then an abscess would almost certainly result from the exploratory puncture, and if an abscess was found then the ultimate outcome would be most doubtful on account of the presence of this supracortical purulent exudate. The fact also that the brain pulsated after the escape of a large quantity of cerebrospinal fluid would have indicated that a subcortical abscess was not present, for the abscess usually causes a marked protrusion of the cortex and the so-called "dry" brain.

Case 85.—Acute severe brain injury with mild signs of increased intracranial pressure; fracture of vault and base of skull. Meningitis. Right mastoiditis; mastoidectomy. Brain abscess; left subtemporal decompression and drainage. Death. Autopsy.

No. 915.—Bella. Forty years. White. Married. School teacher. Ireland.

Admitted November 6, 1917 (6 days after injury), Polyclinic Hospital.

First Operation.—Right mastoidectomy—January 10, 1918—70 days after injury.

Second Operation.—Left subtemporal decompression and drainage—March 18, 1918—138 days after injury and 68 days after first operation.

Died May 8, 1918—188 days after injury and 50 days after second operation.

Family history negative.

Personal history negative.

Present Illness.—While crossing the street, patient was knocked down by an automobile; immediate loss of consciousness; taken in the ambulance to Fordham Hospital, where she remained 6 days; patient was in a condition of severe shock with multiple contusions over head and body and bleeding from the right ear; upon the disappearance of the signs of shock, her general condition improved, and patient was transferred to the Polyclinic Hospital.

Examination upon admission (6 days after injury).—Temperature, 101.6°; pulse, 84; respiration, 26; blood-pressure, 122. Fairly well developed and nourished. Mildly irrational and very irritable. Ecchymosis of both orbits and both mastoid areas. Infected laceration of the scalp over right occipital area. Clotted blood in right external auditory canal; otoscopic
nation reveals a small laceration of lower posterior portion of right
mbrane. Pupils: slightly dilated but react to light normally;
ger than right. Reflexes: patellar, exaggerated but equal;
r Babinski; abdominal reflexes absent. Fundi: retinal
er nasal quadrant of both optic disks blurred—right

more than left. Lumbar puncture—bloody cerebrospinal fluid under hi pressure [illegible] mm.

Treatment.—Expectant palliative. It was hoped that the patient wo be able to take care of the increased intracranial pressure by the na means of absorption and thus an operation be avoided. X-ray (Dr. G. W Wilson)—linear fracture of right occipital bone into right mastoi (Fig. [illegible]). The condition of the patient, however, did not improve and 6 days later, when the signs of increasing intracranial pressure became mo and more marked as exhibited in both fundi by ophthalmoscopic examinatio a subtemporal decompression was advised as the safer method of lower ing the intracranial pressure and to permit a greater ultimate as wel as an earlier recovery; the operation, however, was refused at this time. Patient was, therefore, treated expectantly, but she never regained her normal mental, emotional and physical condition so that she was obliged to remain in the hospital in practically this same condition of mental confusion, irritability and gradual physical weakness for a period of over 2 months. On December 15, 1917 (45 days after the injury), patient complained of stiffness of the neck and seemed more irrational than usual. Definite slight rigidity of the neck with a mild double Kernig sign; lumbar puncture—turbid cerebrospinal fluid under increased pressure (14 mm.); bacteriological report (Dr. Jeffries), "numerous streptococci." Patient was naturally considered "hopeless" and this prognosis was given to the relatives. Fortunately, however, after 2 days, patient improved so much that she became rational again, the rigidity of the neck lessened and the double Kernig sign disappeared; 2 days later, a lumbar puncture revealed clear cerebrospinal fluid under an increased pressure of 12 mm., and the bacteriological report was negative except for an increased cell count of 14 per cubic mm.; no bacteria found. Her condition continued in this slightly improved manner until January 9, 1918, when the patient developed distinct tenderness over the right mastoid area (into which the wide fracture from the occipital protuberance had extended). Doctor J. M. Smith, in consultation, advised a right mastoidectomy, which he performed on January 10, 1918—70 days after injury; much pus and necrosed bone cells removed and the patient made an excellent recovery from this operation so that, as her general condition now improved, she was able to leave the hospital on January 26, 1918—86 days after injury. On February 18, 1918—22 days after discharge—while patient was in the

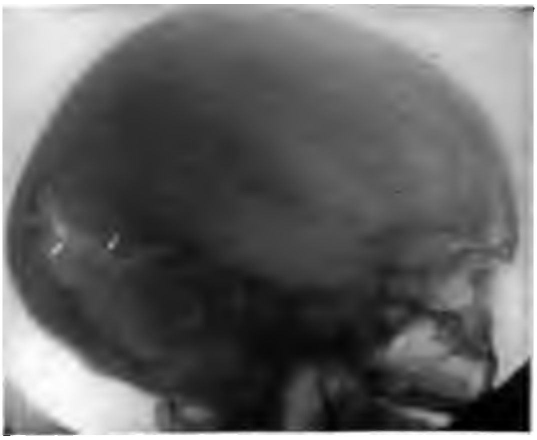


Fig. [illegible].—*Huge linear fracture of right occipital bone extending into the right mastoid area in a patient developing later a purulent mastoiditis and requiring the operation of mastoidectomy.*

out-patient department of the neuro-surgical clinic of the hospital, she suddenly began to have convulsive twitches of the right side of the face, then the right arm, right leg and then a general convulsion occurred which lasted almost one minute; patient was immediately admitted to the hospital, where the following examination was made:

Examination (March 8, 1918—128 days after injury).—Temperature, 99.4°; pulse, 90; respiration, 26; blood-pressure, 128. Perfectly conscious, but in poor condition physically—very much emaciated. Definite weakness of right side of face (cortical type in that right forehead muscles were not involved). Distinct motor and sensory aphasia (patient and relatives all right-handed). Upon percussion, indefinite area of tenderness over the left temporal region. Pupils—left larger than right and reacts sluggishly to light. Reflexes: patellar, very active, right greater than left; right ankle clonus and right Babinski; abdominal reflexes—right absent, left depressed. Fundi: retinal veins enlarged; nasal halves of both optic disks blurred—left possibly more than right. Lumbar puncture: clear cerebrospinal fluid under increased pressure (14 mm.); cell count, 16 cells per c.mm.; no bacteria found.

Treatment.—For fear a localized meningitis was occurring or that a brain abscess was in process of being formed, a left subtemporal exploration and drainage was advised; permission for the operation was not obtained until 10 days later—the patient's condition remaining practically the same.

Second Operation (March 18, 1918—138 days after injury).—Left subtemporal decompression, exploration and drainage: usual vertical incision, bone removed and no complications. Dura thickened, opaque and under moderate tension; upon incising it, a small amount of cerebrospinal fluid escaped, and upon enlarging dural opening, the underlying cortex tended to protrude but did not rupture; upon the cortex, especially in the sulci about the vessels, was a greyish subarachnoid exudate (the result of the former meningitis). The cortex itself did not pulsate; upon exploring the lower fronto-parietal area with a ventricle puncture needle, thick creamy pus welled up through the needle from an abscess cavity situated 4 cm. beneath the cortex in the posterior portion of the left frontal lobe; the double glass tubes for drainage were now inserted, allowing almost 3 ounces of rather thick pus to escape. Bacteriological report (Dr. Jeffries)—"staphylococci." Usual closure with 2 drains of rubber tissue inserted. Duration, 55 minutes. Post-operative notes: Patient made a marked improvement within 10 days after operation; the right facial weakness disappeared, the motor and sensory aphasia improved while the signs of intracranial pressure became less marked; the temperature did not rise above 101°, and there were at no time any signs of meningeal irritation—such as rigidity of the neck, convulsions or a Kernig sign. Patient continued to improve until April 10, 1918 (22 days after the drainage operation), when she gradually became stuporous, paralysis of the right side of the face again appeared and of the cortical type, the left decompression area bulged and the reflexes of the right side of the body again became very active, including a right ankle clonus and a right Babinski; the patient was taken to the operating room, the upper portion of the decompression incision reopened and double glass drainage tubes again inserted into the abscess cavity which was becoming enlarged again by more than left. Lumbar puncture—bloody cerebrospinal fluid under high pressure (18 mm.).

Treatment.—Expectant palliative. It was hoped that the patient would be able "to take care of" the increased intracranial pressure by the normal means of absorption and thus an operation be avoided. X-ray (Dr. G. W. Walton)—"linear fracture of right occipital bone into right mastoid" (Fig. 118). The condition of the patient, however, did not improve and 6 days later, when the signs of increasing intracranial pressure became more and more marked as exhibited in both fundi by ophthalmoscopic examination, a subtemporal decompression was advised as the safer method of lessening the intracranial pressure and to permit a greater ultimate as well as an earlier recovery; the operation, however, was refused at this time. Patient was, therefore, treated expectantly, but she never regained her normal mental, emotional and physical condition so that she was obliged to remain in the hospital in practically this same condition of mental confusion, irritability and gradual physical weakness for a period of over 2 months. On December 15, 1917 (45 days after the injury), patient complained of stiffness of the neck and seemed more irrational than usual. Definite slight rigidity of the neck with a mild double Kernig sign; lumbar puncture—turbid cerebrospinal fluid under increased pressure (14 mm.); bacteriological report (Dr. Jeffries), "numerous streptococci." Patient was naturally considered "hopeless" and this prognosis was given to the relatives. Fortunately, however, after 2 days, patient improved so much that she became rational again, the rigidity of the neck lessened and the double Kernig sign disappeared; 2 days later, a lumbar puncture revealed clear cerebrospinal fluid under an increased pressure of 12 mm., and the bacteriological report was negative except for an increased cell count of 14 per cubic mm.; no bacteria found. Her condition continued in this slightly improved manner until January 9, 1918, when the patient developed distinct tenderness over the right mastoid area (into which the wide fracture from the occipital protuberance had extended). Doctor J. M. Smith, in consultation, advised a right mastoidectomy, which
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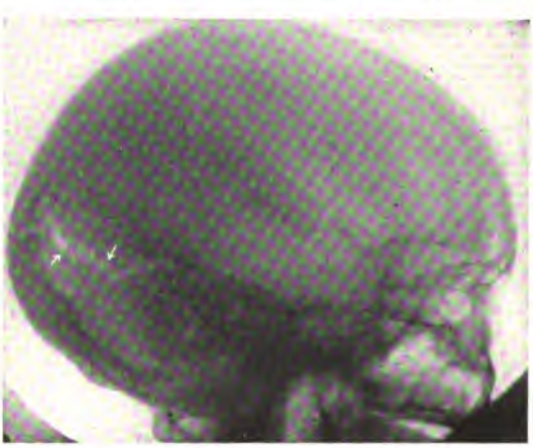

FIG. 118.—Huge linear fracture of right occipital bone extending into the right mastoid area in a patient developing later a purulent mastoiditis and requiring the operation of mastoidectomy.

a low-grade meningitis did result clinically, although the bacteriological report was "numerous streptococci"—and naturally the prognosis was bad. It was most surprising, therefore, to have the patient recover from this diffuse meningitis—only to develop a right mastoiditis and apparently to obtain an excellent recovery, so that the patient was able to leave the hospital as "cured." As a sequela of the former meningitis, Jacksonian convulsions began 128 days after the injury and 83 days after the onset of the meningitis, and it was the left cerebral hemisphere and not the right one contiguous to the line of fracture and the mastoid, which was the site of the abscess with irritation of the overlying cerebral cortex. To have been able to locate and to drain the abscess successfully was most gratifying, and again to have the patient return home as "cured"—the relatives having now been told twice that the patient's condition was practically a hopeless one and each time the patient recovered. However, the abscess had not been successfully drained so that the patient could recover and the general condition becoming weaker and weaker, she died apparently from exhaustion—there being no signs at autopsy of an acute meningitis which was to be expected. From an operative standpoint, the absence of meningitis as the immediate cause of death was gratifying in that this complication is the most common one to be associated with and following the condition of brain abscess.

The autopsy findings revealing multiple lacerations of the anterior surface of the left frontal lobe and directly opposite to the site of the cranial injury and area of contact in the right occipital region, is clearly one of cerebral trauma *au contre-coup*. Even the smaller linear fracture of the orbital plate of the left frontal bone may also be considered as a resulting fracture *au contre-coup*.

Acute Brain Injuries, with and without a Fracture of the Skull, and Their Other Most Frequent Complications.

Brain injuries of any severity occurring in patients over 50 years of age or in patients having a chronic cardio-renal and cardio-vascular condition, and, in fact, any chronic condition which tends to lessen the general resistance of the patient—these injuries must be considered as serious ones, even though the original intracranial damage is little if any at all. If the intracranial lesion is not a serious one in itself, yet the associated stupor and even unconsciousness necessitating a convalescence of days and even several weeks in bed, predispose these patients to many complications other than those directly connected with the cranial injury, such as shock, infection, etc. The most common serious complications of these patients are pneumonia in the elderly; delirium tremens in the alcoholic; mental derangement in the unstable psychically; exacerbations of any chronic organic condition, such as arteriosclerosis, nephritis and diabetes, and thus increasing the danger of an acute cerebral edema; and then, and of greater frequency than is usually supposed, the precipitation of the symptoms and signs of latent lues in its various manifestations, and especially those of cerebrospinal lues and paresis, and it would seem in these latter patients that an active meningeal process was "lighted," so that the former latent condition now appears clinically for the first time; the value of routine physical and neurological examinations with careful laboratory tests of the blood, cerebrospinal fluid and of the urine cannot be overestimated.

Beside these more common complications occurring in brain injuries, there are those patients in whom the condition of brain tumor has been present in a latent condition clinically, and its symptoms and signs suddenly appear following a cranial injury; these cases are most confusing at the time and the lesion of tumor formation is frequently "stumbled upon" at the operation. Cranial injuries of months' or of years' duration may precede and even be in some patients an etiological factor of tumor formation either of the skull, meninges or of the brain itself; this statement, however, cannot be made with certainty.

Acute severe brain injuries complicated by other conditions.

A. *Delirium tremens.*

Case 86.—Acute severe brain injury associated with mild signs of intracranial pressure and a fracture of the base of the skull. No operation. Delirium tremens. Death. Autopsy.

No. 697.—James. Forty years. White. Married. Laborer. U. S.

Admitted October 7, 1916—2 hours after injury—Polyclinic Hospital.

Died October 13, 1916—6 days after injury.

Family history negative.

Personal history negative, except for chronic alcoholism.

Present Illness.—Patient was found lying at the bottom of a stairway; unconscious; brought to the hospital in the ambulance.

Examination upon admission (at least 2 hours after injury).—Temperature, 101.8°; pulse, 118; respiration, 26; blood-pressure, 122. Unconscious; stertorous respiration and signs of an early pulmonary edema—moist râles throughout chest. Marked odor of alcohol on breath. Small contusion and hematoma over right temporal area. Slight bloody discharge from right ear; distinct right mastoid ecchymosis. Pupils slightly enlarged and react sluggishly to light. Reflexes—patellar exaggerated but equal; double exhaustible ankle clonus and double Babinski, Oppenheim and Gordon reflexes; abdominal reflexes absent. Fundi—retinal veins dilated; both retinæ suffused and congested with edematous blurring about the margins of the optic disks. Lumbar puncture—bloody cerebrospinal fluid under increased pressure (approximately 15 mm.).

Treatment.—Expectant palliative; repeated doses of atropine (grains $^1/_{60}$ hypodermically) every 3 hours to control the pulmonary edema; on account of the poor general condition of the patient—the pulse-rate being 118 while the blood-pressure was only 122, it was decided to "watch" the patient carefully in the hope that the condition would improve. Within 24 hours, the temperature had descended to 100°, the pulse to 104 and the respiration to 24, while the blood-pressure had risen to 136 and patient had become semiconscious; the pulmonary râles had almost disappeared and as the signs of the increased intracranial pressure had not become more marked, it was believed that the patient would recover without an operation being necessary—that is, the intracranial hemorrhage and the cerebral edema would be absorbed by the natural means of excretion from the cerebrospinal canal. On the second day after admission, however, the patient suddenly developed a fine and then a coarse tremor of both hands, became mentally confused and irrational, had definite hallucinations of sight—the typical signs of beginning delirium tremens. In spite of active treatment for this condition (repeated lumbar punctures, the administration of alcohol, morphia, hot packs, etc.), the patient steadily became worse and finally died from the typical condition of "wet" brain, resulting from the so-called delirium tremens—6 days after injury.

Autopsy.—Linear fracture extended from right parietal crest vertically downward through the squamous portion of the right temporal bone, through the posterior portion of left tympanic membrane along the petrous bone to within one inch of its apex; no other fracture found (Fig. 120). Much blood and cerebrospinal fluid, particularly in the middle and posterior fossæ. Brain itself very "wet" and edematous, but no cortical hemorrhage or laceration. Ventricles negative.

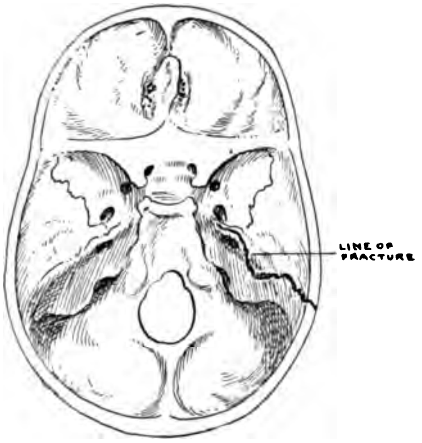

FIG. 120.—Wide linear fracture extending through the right petrous bone in a patient who would probably have recovered if the complication of delirium tremens had not occurred.

Remarks.—If this complication of delirium tremens had not occurred, it seems that this patient would have recovered. It was a mistake not to have administered alcohol to this patient, either by mouth or by rectum, so that the onset of acute alcoholism could have been avoided if possible; all patients accustomed to the daily use of alcohol, and particularly having a cranial injury of any severity, should be given daily at least one-half their usual allowance of alcohol and thus the danger of delirium tremens be lessened. It is a well-known observation, however, that patients who are at all alcoholic withstand very poorly the effect of head injuries owing to the fact that a cerebral edema occurs much more easily in these patients, so that, even if alcohol were given after their admission to the hospital, the cerebral edema precipitating delirium tremens would frequently occur in spite of this precaution. Chronic alcoholics are very poor risks, whether an operation is performed or not, and there is no one factor which increases the mortality of brain injuries more than that of chronic alcoholism.

The clinical picture of this patient upon admission having a temperature of 101.8°, pulse 118, respiration 28, blood-pressure 122, would indicate that the patient was in the condition of mild shock with the lowered blood-pressure and the increased pulse-rate, while the mild temperature was due to the early signs of pulmonary edema associated with chronic alcoholism; this opinion would tend to be substantiated by the observation that 24 hours later the temperature descended to 100° and not to subnormal and the pulse to 104, while the blood-pressure increased to 136, synchronous with the lessening of the pulmonary edema.

CASE 87.—Acute severe brain injury associated with mild signs of high intracranial pressure and a fracture of the base of the skull. No operation. on December 2, 1914—29 days after injury.

No. 169.—John. Thirty-two years. White. Single. Storekeeper. Greece. Admitted November 3, 1914—1½ hours after injury, Polyclinic Hospital. Transferred to Bellevue Hospital November 6, 1914; discharged "well," on December 2, 1914—29 days after injury.

Family history negative.

Personal History.—As the result of a profitable sale of his store, patient has been drinking heavily during the past 6 weeks and his friends say "his only hope is in his losing his money."

Present Illness.—While intoxicated, patient was "black-jacked" by thugs who obtained the balance of patient's money; unconscious; brought to the hospital in the ambulance.

Examination upon admission (90 minutes after injury).—Temperature, 99°; pulse, 88; respiration, 24; blood-pressure, 130. Semiconscious but can be easily aroused; mildly delirious and behaves foolishly. Marked tremor of both hands. Contusion and ecchymosis over left eye and left lower jaw. Slight bleeding and discharge of bloody cerebrospinal fluid from left ear; distinct left mastoid ecchymosis. Pupils equal and react normally. Reflexes negative. Fundi negative. Lumbar puncture—blood-tinged cerebrospinal fluid under moderate pressure (approximately 13 mm.).

Treatment.—Expectant palliative; small amounts of alcohol given to the patient and the routine prophylactic treatment for chronic alcoholism. In spite of this treatment, however, patient progressively became worse in that the hallucinations became more realistic to him, associated with an acute delirium, requiring restraint, so that 3 days after admission, it was considered advisable that the patient be transferred to the alcoholic ward of Bellevue Hospital, where he could be treated more competently for this complication. His record at Bellevue was that of a typical case of delirium tremens; the recovery was uneventful and the patient was discharged as "cured"—29 days after injury.

Last Examination (May 20, 1917—31 months after injury).—Patient no longer drinks and has no complaints; he has acquired a small grocery store from his savings during the past 2 years. Hearing—no impairment of either ear can be elicited; otoscopic examination of left ear negative; air conduction greater than bone conduction. Reflexes negative. Fundi negative.

Remarks.—This patient, being a youthful adult, had a greater chance of recovery both from the cranial injury and from the attack of acute alcoholism than if he had been 15 years older and more addicted to the chronic use of alcohol—then, the prognosis as to life would have been much more doubtful. The fracture of the skull through the ear and thereby allowing the intracranial hemorrhage to escape was most fortunate for him in that the increased intracranial pressure was undoubtedly lessened and thus enabled him, not only to recover much more easily, but much more rapidly and without a cranial operation.

The absence of permanent impairment of hearing in many of these patients having fractures of the skull which have extended through either ear is very interesting; it occurs in possibly 20 per cent. of these patients.

It would seem that patients of middle age and older who are addicted to the daily use of alcohol for over a period of years and if only in moderation, that there is an increased amount of cerebrospinal fluid normal to these patients—that is, a mild degree of the so-called "wet" brain. Any severe illness, and particularly a cranial injury, which upsets their body metabolism and in any way diminishes their daily supply of alcohol, then these patients are very liable to develop an acute condition of cerebral edema and thus the typical onset of delirium tremens occurs. The mild toxic condition resulting from this daily continued use of alcohol lessens the power of resistance of these patients and they are much more liable to succumb to any severe mental and physical strain. They are always "bad" operative risks, including the greater danger of anesthetic complications, and the prognosis must be very guarded, especially following cranial injuries.

B. *Brain tumor.*

CASE 88.—Acute severe brain injury associated with definite signs of an increased intracranial pressure and with an operative fracture of base of skull. Bilateral decompression and drainage. Death. Autopsy; midbrain sarcoma.

No. 794.—James. Forty years. White. Married. Laborer. U. S.

Admitted February 29, 1917—10 days after operative injury. Polyclinic Hospital.

First Operation (March 2, 1917—2 days after admission and 12 days after operative injury).—Left subtemporal decompression and drainage.

Second Operation (March 4, 1917—2 days after first operation and 14 days after injury).—Right subtemporal decompression and drainage.

Died March 8, 1917—4 days after second operation and 18 days after injury.

Family history negative.

Personal History.—During the past 6 months, patient has been treated at an Eye and Ear Hospital for chronic inflammation of the ethmoid and sphenoid sinuses and also for a chronic right otitis media; several operations had been performed upon both the ethmoid and sphenoid sinuses and the right middle ear "cleaned out." Patient was improved for several weeks and then his symptoms and signs returned—particularly the frontal headache, the purulent discharge from the nose and the spells of dizziness.

Present Illness.—On February 19, 1917 (10 days before admission), patient was operated upon and the sphenoidal cells opened and evacuated; immediately after this operation, patient became stuporous, complained of severe bifrontal headache and was mildly irrational; 3 days before admission, patient suddenly became blind in both eyes, stupor increased and the patient complained of an increasing deafness in both ears. Transferred to the hospital in the ambulance.

Examination upon admission (10 days after the sphenoidal sinus operation).—Temperature, 99.8°; pulse, 72; respiration, 20; blood-pressure, 142. Semiconscious but can be easily aroused. Slight purulent discharge from the right ear. Thin bloody discharge from both nostrils. Totally blind in both eyes—not even light perceived; external ocular muscles all paralyzed (ophthalmoplegia externa) so that both eyeballs cannot be moved. Hearing of both ears apparently impaired. Slight right facial weakness of the cortical type. Pupils moderately dilated and do not react to light. Reflexes—patellar can be elicited with difficulty and are apparently equal; no ankle clonus nor Babinski; abdominal reflexes absent. Fundi—retinal veins dilated; both nasal and temporal margins blurred and indistinct but no papilledema or measurable swelling of either optic disk. Lumbar puncture—clear cerebrospinal fluid under high pressure (18 mm.); cell count—6 cells per c.mm. and no bacteria present. No weakness of extremities and no sensory impairment could be elicited. X-ray "negative" (Doctor W. H. Stewart).

Treatment.—On account of the increased intracranial pressure and the appearance of a right facial palsy of the cortical type, a left subtemporal decompression and exploration was advised; consent for the operation could not be obtained until 40 hours later, and as the condition of the patient was practically the same as at the preceding examination, the operation was now performed.

First Operation (2 days after admission and 12 days after operative injury).—Left subtemporal decompression and exploration: usual vertical incision, bone removed and no complications. Dura very tense and slightly bluish; upon incising it, blooody cerebrospinal fluid spurted to a height of 5 inches; upon enlarging dural opening, the underlying cerebral cortex tended to protrude under high tension; it did not rupture, however, but only slight pulsation could be observed. Cortex contained several small punctate hemorrhages and much subarachnoid blood and cerebrospinal fluid escaped. An attempt to tap the left lateral ventricle not successful and careful probing of the left frontal lobe, left parietal lobe and left temporosphenoidal lobe for brain tumor was negative. Usual closure with 2 drains of rubber tissue inserted. Duration, 45 minutes.

Post-operative Notes.—The stupor of patient improved for 24 hours and then he gradually became semiconscious again, the decompression area bulged and the signs of an increasing intracranial pressure reappeared; a right subtemporal decompression was now considered advisable both to lessen the intracranial pressure and in the hope that a tumor or abscess could be located in the right hemisphere, or if it was merely a cerebral edema, then its resulting increased pressure could be successfully lessened. (The sudden impairment of vision could not be explained as well as the ophthalmoplegia externa, unless the condition was a mid-brain tumor which by a sudden enlargement had directly compressed both optic nerves and the ocular nerves.)

Second Operation (2 days after first operation and 14 days after injury).—Right subtemporal decompression and exploration: usual vertical incision, bone removed and no complications. Dura very tense and upon incising it, slightly blood-tinged cerebrospinal fluid welled through dural opening under high pressure—revealing a very "wet," edematous cortex which tended to protrude but did not rupture, owing to the escape of a large amount of cerebrospinal fluid, so that the cortex pulsated normally at the end of the operation. Right lateral ventricle punctured, allowing clear cerebrospinal fluid to escape under moderate pressure. Careful exploratory punctures of the right frontal, right parietal and right temporo-sphenoidal lobes were all negative. Usual closure with 2 drains of rubber tissue inserted. Duration, 50 minutes.

Post-operative Notes.—Patient made a marked improvement during the first few days after operation in that he became conscious, was able to talk rationally and he no longer complained of headache; the vision, however, did not improve, and on the third day post-operative, the temperature quickly ascended to 105°, pulse to 138 and respirations to 36; both decompression areas bulged tensely, while the patient became more and more stuporous. Lumbar puncture revealed clear cerebrospinal fluid under moderate increase of intracranial pressure (13 mm.); cell count was 10 cells per c.mm.; the condition of patient rapidly became worse, pulmonary edema occurred, and in spite of vigorous stimulative measures and the use of atropine, the patient became weaker and weaker and finally died—4 days after second operation and 18 days after injury.

Autopsy.—Upper portion of sphenoidal plate had been fractured just in front of the anterior clinoid processes and in close proximity to both optic nerves (Fig. 121). Much free subdural blood in both the anterior and middle fossæ, while an extensive blood-clot lay upon the inferior surface of both frontal lobes. In the mid-brain superimposing the sella turcica and both optic nerves, which were being directly compressed from above downward, was a hard fibrous tumor—the size of an orange. (Pathological report (Doctor Jeffries)—''spindle-cell sarcoma''); this tumor mass lay beneath and slightly anterior to the third ventricle and thus the lateral ventricles had not been blocked; this was very fortunate for the patient in that an internal hydrocephalus did not develop. Posterior fossa negative. Ventricles negative.

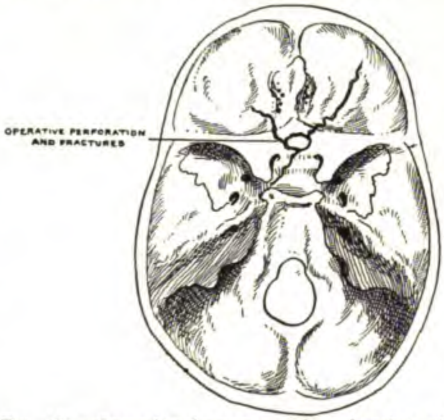

FIG. 121.—Operative fractures and perforation of median portion of sphenoidal bone in a patient dying from a large sarcoma of the mid-brain and superimposed upon the sella turcia—the cause for his symptoms and signs.

Remarks.—This is another case—and they are very common—of a mid-brain tumor simulating sphenoidal sinus disease; the right chronic otitis media and the chronic sinusitis both tended to obscure the diagnosis, and it was not until a sudden complete loss of vision occurred associated with the ophthalmoplegia that a mid-brain lesion was suspected. In attempting to perform the sphenoidal sinus operation, the upper and posterior bony wall had been accidentally fractured with a resulting subdural hemorrhage from the adjoining venous sinus which fortunately became thrombosed early and did not extend to the other side. It was then, and apparently for the first time, that the signs of an increased intracranial pressure occurred necessitating the decompression operations.

The tumor was of such a large size and so situated in the mid-brain that an operative removal of it could not have been attempted successfully; if it had been smaller, it possibly could have been removed by the lateral approach—either by retracting the temporo-sphenoidal lobe upward, or, if necessary, the removal of the right temporo-sphenoidal lobe which permits an excellent exposure of mid-brain tumors and facilitates their successful enucleation, especially if a bilateral decompression has been performed so that the brain can be "dislocated," and thus a better surgical exposure be obtained.

The immediate cause of death in this patient was evidently an acute medullary edema precipitated by the general lowered resistance of the patient and the intracranial lesions. How this patient escaped a purulent meningitis through infection of the operative fracture of the base of the anterior fossa is difficult to explain in the presence of a purulent sphenoidal sinusitis. The sudden onset of blindness was probably due to an acute edema of the tumor itself or to small hemorrhages into the tumor itself (as disclosed upon sectioning the tumor), and thus both optic nerves at the chiasm were markedly compressed; naturally, if this compression could not have been relieved and if the patient had lived for a period of weeks, the ophthalmoscope would have revealed definite signs of a primary optic atrophy. If this compression of both optic nerves had not been so great, then a lateral or horizontal hemianopsia would have occurred and this observation would have facilitated the diagnosis very much indeed; the total ophthalmoplegia should have emphasized the localization of the lesion more than it did, but at the time it was considered more of a retro-orbital or posterior orbital condition due to the inflammatory lesion of the sinuses. The value of post-mortem examinations not only to the medical attendant but to future patients, could not be more highly emphasized and appraised than in the case of this patient; more is frequently learned from mistakes than from successes.

The differential diagnosis before operation upon this patient was most perplexing: the history tended to indicate a brain abscess and meningitis, particularly associated with the sphenoidal sinusitis and the purulent otitis media; the increased intracranial pressure, however, of a height of 18 mm. and yet the cerebrospinal fluid clear and with a cell count of only 6 cells per c.mm. and no bacteria present, tended to the diagnosis of brain tumor. It was most surprising, therefore, to find a subdural hemorrhage at operation, and it was then that the sphenoidal sinus operation was considered as the probable cause of the intracranial condition—being similar to a brain injury following a fracture of the skull, while the impairment of vision and ophthalmoplegia were due to a local injury at the time of the operation itself. The autopsy findings were indeed most instructive.

Case 89.—Acute severe brain injury associated with definite signs of an increased intracranial pressure and with a fracture of the base of the skull. Left subtemporal decompression and drainage. Death. Autopsy; glioma of left temporo-sphenoidal lobe.

No. 977.—Clara. Fifty years. White. Married. Housework. U. S.

Admitted May 6, 1918—21 days after injury—Polyclinic Hospital. Referred by Doctor Adolph Reich.

Operation (May 9, 1918—3 days after admission and 24 days after injury). Left subtemporal decompression and drainage.

Died May 10, 1918—26 hours after operation and 25 days after injury.

Family history negative.

Personal history negative.

Present Illness.—Twenty-one days before admission, patient was knocked down by a street car; immediate loss of consciousness and multiple contusions of both arms and legs; slight bleeding from left ear; patient was taken to her home in a taxicab, but she had so recovered on the way home that she walked into her house and did not go to bed. Definite impairment of speech, however, immediately appeared—unable to say words that she wished to; complained of headache but otherwise "fairly well." Seven days after injury, patient was obliged to be restrained in bed on account of her becoming irrational, staggered as though drunk and could not talk coherently; complained of "bad odors," and she could not be convinced that they were not present. On account of the increasing headache, the patient was brought to the hospital.

Examination upon admission (21 days after injury).—Temperature, 100°; pulse, 84; respiration, 24; blood-pressure, 136. Mildly irrational. Definite weakness of entire right side of body; also, a distinct impairment of sensation both to light-touch and to pain over the entire right side of the body. (This last observation could not be confirmed owing to the mental condition of the patient.) Otoscopic examination—laceration of lower posterior quadrant of left tympanic membrane. Pupils—left larger than right and reacts sluggishly to light. Reflexes—patellar not increased though right greater than left; no ankle clonus but double Babinski, Oppenheim and Gordon reflexes; abdominal reflexes—right absent, left depressed. Fundi—retinal veins enlarged; nasal halves of both optic disks blurred—left more definitely than right, but no measurable swelling of the disks. Lumbar puncture—straw-colored cerebrospinal fluid under high pressure (20 mm.); pathological report (Doctor Jeffries)—"numerous broken-down red corpuscles." Urine examination—slight trace of albumen; occasional hyaline cast.

Treatment.—Expectant palliative. The condition of the patient, however, became worse daily in that the signs of pressure became more marked in the fundi, and the weakness of the right arm and right leg progressed to the extent of almost a complete paralysis of them; the increasing stupor of the patient with a gradual lowering of the pulse-rate made a subtemporal decompression imperative, if the patient was to be given a chance to recover; the pulse did not descend below 70, however, while the temperature ascended to 102.6°.

Operation (24 days after injury).—Left subtemporal decompression (no anesthesia being necessary): usual vertical incision, bone removed and no complications. Dura very tense and upon incising it, very little cerebrospinal fluid escaped but the underlying cortex protruded through the small dural opening under high pressure, ruptured and brain tissue oozed out; an attempt was made to puncture the left lateral ventricle but unsuccessfully. Upon enlarging dural opening therefore owing to the high cerebral tension, the entire underlying cortex ruptured, permitting gelatinous grumous degenerated nerve tissue and débris to extrude and revealing a cavity in the left temporo-sphenoidal lobe—the size of an orange. The condition of the patient becoming rapidly worse, a hurried closure was made with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—The condition rapidly became worse; patient did not regain consciousness and within 8 hours the temperature had ascended to 106°, pulse to 140 and respiration to 42, while the blood-pressure had descended to 88. Patient died 26 hours after operation—from a condition typical of an acute medullary edema.

Autopsy.—Small fracture of left petrous bone extending through left middle ear (Fig. 122). Small amount of straw-colored cerebrospinal fluid in both anterior and middle fossæ. Anterior surface of right frontal lobe contused and lacerated; also the tip of the right sphenoidal lobe. Occupying the entire left temporo-sphenoidal lobe was a cavity, the size of an orange, filled with degenerated brain tissue. (Pathological report (Doctor Jeffries)—"degenerated gliomatous tissue.") Left ventricle had been collapsed and forced toward the median line. Posterior fossa negative.

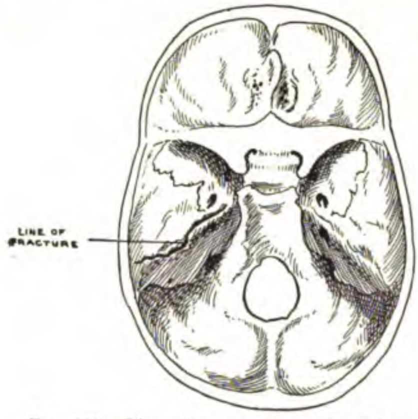

FIG. 122.—Linear fracture of left petrous bone, following a "bump" upon the head of a patient having a large gliomatous tumor of the left temporo-sphenoidal lobe.

Remarks.—The clinical syndrome of this patient is very instructive; although this gliomatous tumor must have existed for a period of at least several months before the injury, yet there was no history of any complaints at all—the patient being considered in the best of health. It is conceivable that the cranial injury, besides causing a contusion and laceration of the right frontal and right sphenoidal lobe by *contre-coup*, had also at the same time caused a hemorrhage into the tumor mass itself in the left temporo-sphenoidal lobe or had at least precipitated the degeneration of the tumor mass itself by causing an acute cerebral edema, and thus introducing and then accentuating the symptoms and signs of a lesion of the left temporo-sphenoidal lobe (the patient being right-handed and all of his blood relatives being right-handed): the disagreeable odors and the state of unreality—the relatives stated that before coming to the hospital, the patient complained of dreaming, and yet while she lay dreaming she knew that the dreams were not true and that these dreamy states of unreality persisted until she become irrational; no impairment of taste had been elicited nor any impairment of the hearing, however; the neighborhood signs of motor aphasia and both a motor and sensory involvement of the right side of the body were also observed. It is possible if the cranial injury had not occurred th patient could have lived a number of months and even a perio year or more before the symptoms and signs of the tumor mas been observed. If it had not been for this complicat operative recovery of the patient from the brain injury would have been very possible. Unfortunately, this patient had been allowed to progress to such a condition of general physical exhaustion, that a medullary edema occurred chiefly on account of the lessened resistance of the patient having an intracranial lesion of this character.

It was a very confusing coincidence that the fracture of the skull in this patient passed through the left middle ear rupturing the left tympanic membrane, to be followed by definite symptoms and signs of a left temporosphenoidal lesion; the unusually high intracranial pressure of 20 mm. at a period 3 weeks after the cranial injury is most unusual, and an intracranial tumor might have been suspected at this time as being the underlying cause of the patient's condition; at the time, a possible large left extradural hemorrhage was considered as being the cause of the condition.

The inability to puncture the left lateral ventricle before the dura was widely opened and thus prevent an operative damage to the underlying cerebral cortex owing to the very high intradural pressure, is explained by the autopsy findings in which the left lateral ventricle was found to be forced downward and toward the median line and also collapsed by the pressure of the tumor mass in the left hemisphere; a lumbar puncture might have been attempted at this time and by this means the intradural pressure lessened so that no operative damage to the underlying cerebral cortex would have occurred.

C. *Mental derangements.*

CASE 90.—Acute severe brain injury associated with high intracranial pressure and with a fracture of the base of the skull. Left subtemporal decompression and drainage. Mental and emotional impairment. Recovery.

No. 145.—Edwin. Fifty-eight years. White. Married. Broker. U. S.

Admitted December 16, 1914—70 minutes after injury—Flower Hospital. Referred by Doctor G. R. Satterlee.

Operation (December 19, 1914—3 days after injury).—Left subtemporal decompression and drainage.

Discharged January 29, 1915—40 days after injury.

Family history negative.

Personal History.—During the past 6 years, patient has had 2 very severe attacks of acute nephritis requiring a hospital residence of over 4 months each time; marked arteriosclerosis. Patient has been considered eccentric and emotionally unstable for years.

Present Illness.—While descending the subway stairs, patient fell headlong to the bottom; immediate loss of consciousness; taken to the Flower Hospital in ambulance; patient remained unconscious and in severe shock; profuse bleeding from the left ear.

Examination (66 hours after admission. Consultation with Doctor Satterlee).—Temperature, 100°; pulse, 68; respiration, 20; blood-pressure, 156. Semiconscious and exceedingly restless. Marked arteriosclerosis—radial arteries hard and tortuous and temporal arteries similarly affected. Small lacerations of the scalp posterior to the external angular process of left bit. Clotted blood in left auditory canal; otoscopic examination reveals a ation of posterior half of left tympanic membrane; extensive left mastoid ecchymosis. Pupils equally contracted and do not react to light. Reflexes—patellar exaggerated, right more than left; exhaustible ankle clonus and right Babinski; abdominal reflexes present and equal. Fundi—retinal veins dilated; nasal margins of both optic disks and nasal half of left optic disk blurred by edema. Lumbar puncture—bloody cerebrospinal fluid under high pressure (approximately 19 mm.). Urine examination—trace of albumen; many hyaline and finely granular casts.

Treatment.—Although the patient was not considered a good operative risk owing to the condition of arteriosclerosis and chronic nephritis, yet it was considered the safer procedure to lessen the increasing intracranial pressure by the operation of subtemporal decompression and drainage than to allow the patient to attempt the absorption of the intracranial hemorrhage and cerebral edema by natural means—that is, the risk of an operation was less than the effect of a prolonged increase of the intracranial pressure.

Operation (68 hours after admission).—Left subtemporal decompression: usual vertical incision, bone removed and no complications; much watery fluid (subcutaneous edema) throughout the tissues of the scalp and temporal muscle. Dura exceedingly tense and upon incising it, slightly blood-tinged cerebrospinal fluid welled up through the dural opening, exposing a very "wet," swollen, edematous brain, which did not rupture on account of the rapid escape of the cerebrospinal fluid. No cortical hemorrhage nor laceration visible. At end of operation, the cortex pulsated normally. Usual closure with 2 drains of rubber tissue inserted. Temporary sterile dressing applied. The small depressed fracture just posterior to the left angular process of the orbit was now removed; the dura had not been torn and was naturally not opened. Scalp sutured loosely with one drain of rubber tissue inserted. Duration, 1 hour.

Post-operative Notes.—Within 12 hours, the patient became more conscious and the pulse ascended to 80; within 36 hours, the signs of an increasing intracranial pressure, as revealed by the ophthalmoscope, were lessened so that there remained only a slight blurring of the lower nasal quadrant of both optic disks. Patient made an excellent operative recovery, although he was mildly irrational during the entire period of hospital residence; owing to this emotional instability which was greater than before the injury, it was considered advisable to transfer him to Bloomingdale Hospital, White Plains, for a period of 3 months, in order that the patient should be in as ideal conditions as possible for the recovery of both mental and emotional stability.

Examination upon transfer (40 days after injury and 37 days after operation).—Temperature, 99°; pulse, 88; respiration, 24; blood-pressure, 160. Mildly irrational and excitable. Decompression area flush with surrounding scalp and pulsates normally. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi negative. Urine exami tion—trace of albumen; many hyaline and granular casts.

The patient remained in Bloomingdale Hospital for a period of months; his condition had so improved that he was able to r family and after the summer's vacation, he was able to retu work in October, 1915—10 months after the injury. H time was considered as normal mentally and emotion

Examination (May 18, 1916—29 months after injury).—No complaints; patient is rather effusive, however—wife states that he always was so. Decompression area depressed and pulsates normally. Hearing of left ear impaired; bone conduction is greater than air conduction. Reflexes active, but otherwise negative. Fundi negative. Urine examination—trace of albumen; many finely granular and hyaline casts.

Patient continued in this condition until November, 1917, when he died following an attack of pneumonia.

Remarks.—It is doubtful whether this patient could have recovered unless the lessening of the increased intracranial pressure had not been obtained by the cranial decompression, and such an excellent recovery of the patient's former normality (for him) would not have been probable, if the increased intracranial pressure had been allowed to continue for an indefinite period of time; handicapped as the patient was by arteriosclerosis and chronic nephritis, it is difficult to conceive of the patient approximating his former condition to any degree. It is too much to expect that his mental and emotional condition could be improved by a cranial injury and such a benefit cannot be conceived, but if a patient is thus already impaired, the hope should be that the impairment is not increased by the injury, and this fortunate result can frequently be obtained by the early relief of the increased intracranial pressure resulting from the cranial injury.

The presence of a watery edema in the subcutaneous tissues, as disclosed at operation and in the absence of an adjacent fracture of the skull with a tear of the underlying dura (and thus indicating the escape of the cerebrospinal fluid), is usually a bad prognostic sign in that this subcutaneous edema usually occurs when the resistance of the patient is very much lowered and particularly in patients having arteriosclerosis and chronic nephritis. The great danger of a severe cerebral edema resulting from the cranial trauma in these patients and thus the risk of the early onset of an acute medullary compression and edema must always be feared, so that the early decompression and drainage is more advisable in these patients than in the ones who are in a better condition of resistance.

A fracture of the skull presumably passed through the left mastoid area into the petrous portion of the left temporal bone, since there was a definite left mastoid ecchymosis and a profuse discharge of blood through a laceration of the left tympanic membrane; no cerebrospinal fluid, however, was observed in this aural discharge and therefore, it cannot be stated with absolute certainty that a fracture of the base of the skull did occur; no fracture of the left squamous bone, was observed at operation, but that frequently happens even when there is a fracture of the left petrous bone; it is a rare occurrence, however, for the tympanic membrane to be ruptured indirectly by a cranial injury in the absence of a fracture of the adjacent temporal bones. The slightly depressed fracture just posterior to the angular process of the left orbit was of practically no importance in that the under-
ig dura was intact and the depression was of such slight degree that it is
if any impairment or irritation of the underlying cerebral cortex
oduced by it. It is not the question of the fracture of the
e, but the presence or not of a marked increase of

the intracranial pressure and that was well "taken care of" by the subtemporal decompression and drainage.

CASE 91.—Acute severe brain injury associated with signs of high intracranial pressure and with a fracture of the base of the skull. Right subtemporal decompression and drainage. Mental and emotional impairment. Excellent recovery.

No. 971.—Frederick. Twenty-six years. White. Single. Clerk. U. S.

Admitted November 7, 1917—45 minutes after injury—Nassau Hospital, Mineola. Referred by Doctor G. F. Cleghorn.

Operation (November 8, 1917—32 hours after injury).—Right subtemporal decompression and drainage.

Discharged January 6, 1918—58 days after operation.

Family history negative.

Personal history negative.

Present Illness.—Following an automobile collision, patient was thrown headlong to the road; immediate loss of consciousness; taken to the hospital in the automobile. Upon admission, patient was profoundly unconscious and in severe shock—the temperature being 97.8°, the pulse 118, while the blood-pressure was only 106; profuse bleeding from right ear and multiple contusions. Vigorous shock measures instituted so that the patient quickly recovered from the condition of shock within a period of 12 hours, and it was only then that the signs of an increasing intracranial pressure appeared.

Examination (30 hours after injury; consultation with Doctor Cleghorn).—Temperature, 99.8°; pulse, 66; respiration, 18; blood-pressure, 132. Semiconscious and very restless—confused as to time, place and personality; restraint is required to keep the patient in bed. Slight bleeding from the right ear; distinct right mastoid ecchymosis. Multiple contusions over body and extremities. Pupils enlarged—right more than left and reaction to light sluggish. Reflexes—patellar very active but equal; double ankle clonus and double Babinski; abdominal reflexes absent. Fundi—dilated retinal veins; nasal halves and temporal margins of both optic disks obscured by edema. Lumbar puncture—bloody cerebrospinal fluid under high pressure (approximately 20 mm.).

Treatment.—An immediate right subtemporal decompression advised to lower the intracranial pressure and to lessen the cortical irritation for fear of not only an acute medullary compression and medullary edema but the great danger of the cortical irritation becoming so great that epileptiform seizures would occur.

Operation (32 hours after injury).—Right subtemporal decompression: usual vertical incision, bone removed and no complications; fibres of temporal muscle were hemorrhagic and therefore a fracture of the unde
squamous bone was found extending obliquely downward toward the
nal auditory canal. No extradural hemorrhage but the dura itself
tense and bluish, and upon incising it, bloody cerebrospinal fluid sp
height of 2 inches; upon enlarging dural opening, a layer of s
hemorrhage of 1 cm. in thickness was exposed and evacuat
cortex congested and edematous but no cortical hemorrh

observed. At the end of the operation, the cortex pulsated normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Within 12 hours after operation, the signs of increased intracranial pressure lessened and the general condition of the patient improved; patient remained, however, in a highly excitable condition, requiring restraint, and these signs of cortical irritability persisted for over 7 weeks; during this period, he remained disoriented as to time, place and personality, babbled continuously—both sense and nonsense; the physical condition, however, had so improved that the reflexes had become negative and the fundi normal; the decompression area was flush with the surrounding scalp and pulsated normally. This mental condition was apparently due to the edematous condition of the brain, so that when this cerebral edema was absorbed at the end of 7 weeks, he made an immediate improvement and was able to be discharged on January 6, 1918—59 days after injury. X-ray report —"wide irregular fracture of occipital bone extending forward toward right mastoid bone; oval bone defect of right decompression" (Fig. 123).

Last Report (September 18, 1919—22 months after injury).—Patient has regained his former good health and is working daily; no complaints.

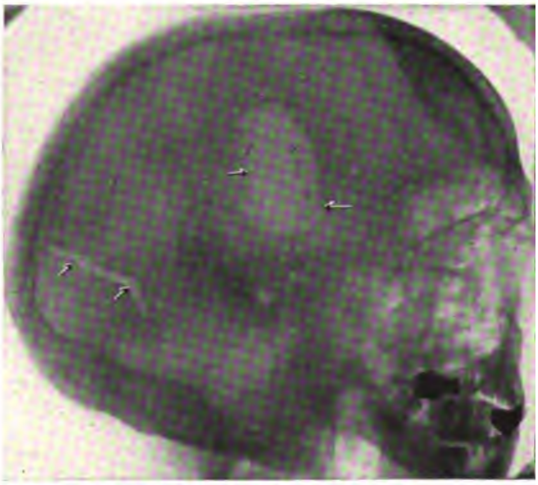

FIG. 123.—Lateral röntgenogram showing a linear fractur of the right occipital bone extending forward toward the righ mastoid area. The bony defect of a right subtemporal decompression, with three silver clips clamping the dural vessels, ca be seen. Patient has made an excellent recovery.

Remarks.—It is rather unusual for cranial injuries to produce such a persistent cerebral edema and its resulting mental and emotional impairment as in this patient, unless the patient is beyond middle age and particularly if the patient is alcoholic or arteriosclerotic; these latter patients permit a chronic cerebral edema to occur more easily and to persist for varying periods of time, but it is rare for this condition to appear in youthful adults and especially in those of temperate habits. If it required a period of almost 2 months for this patient to recover his mental and emotional equilibrium even with a marked lessening of the intracranial pressure and consequent drainage of the excess cerebrospinal fluid by means of an operation, it is easy to conceive that, without an operation, not only would the recovery of life have been doubtful but even if such a recovery of life should occur, then the great risk of future mental and emotional impairment would have been very probable. Naturally, a longer period of time must elapse in the case patient before we can estimate more accurately whether there has permanent damage or not.

Case 92.—Acute severe brain injury associated with mild signs of intracranial pressure and with a fracture of base of skull. No operation. Delirium tremens and cerebrospinal lues. Luetic treatment. Improved.

No. 721.—Richard. Forty-eight years. White. Single. Laborer. U. S.

Admitted November 11, 1916—50 minutes after injury—Polyclinic Hospital.

Transferred to Bellevue Hospital November 26, 1916—15 days after admission.

Family history negative.

Personal History.—No luetic history obtainable; chronic alcoholism, especially during the past 10 years.

Present Illness.—While crossing the street, patient is said to have fallen, striking his head against the asphalt pavement; an immediate general convulsion occurred, and it is not known whether the convulsion was the cause of the fall or the cranial injury was the cause of the convulsion; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (50 minutes after injury).—Temperature, 99°; pulse, 88; respiration, 26; blood-pressure, 152. Well-developed and nourished. Unconscious and having general convulsions—almost the condition of status epilepticus. No contusion of scalp ascertained. Profuse bleeding and discharge of cerebrospinal fluid from the left ear; slight left mastoid ecchymosis. Pupils: equally contracted and reaction to light sluggish; slight irregularity of the pupillary outline. Reflexes—patellar very much exaggerated but equal; double ankle clonus and double Babinski; abdominal reflexes absent. Fundi—retinal veins dilated; both retinæ congested and suffused and of a brick red appearance; both nasal and temporal margins rather blurred and indistinct. Lumbar puncture—bloody cerebrospinal fluid under increased pressure (16 mm.).

Treatment.—Expectant palliative. Within 12 hours after admission, the patient became conscious but was very irrational and mildly maniacal, requiring restraint; marked tremor of both hands appeared, hallucinations of vision and the typical picture of delirium tremens of alcoholism developed; vigorous treatment instituted and fortunately at the end of 4 days the patient had made such a marked improvement that restraint was no longer required. Pathological report of the cerebrospinal fluid was now registered as being 4 plus; blood was also 4 plus, and therefore active luetic treatment was begun immediately. The signs of the increased intracranial pressure became less marked at the end of 8 days, but the patient still remained in a mildly irrational condition—mentally confused, disoriented as to time, place and personality, and emotionally unstable—crying at times and then very irritable. On account of the difficulty of treating a patient so affected in a general surgical ward, the patient was now transferred to Bellevue Hos

Examination upon transfer (to Bellevue Hospital—15 days after a
sion).—Temperature, 99.2°; pulse, 84; respiration, 24; blood-pressur
Rather emotional, and mentally confused. Otoscopic examination r
a small laceration of the upper posterior quadrant of the left tympa
brane; hearing tests could not be made on account of the mental c
the patient. Pupils—of normal size and reaction; pupillary

irregular. Reflexes—patellar very active but equal; double exhaustible ankle clonus and tendency to double Babinski; abdominal reflexes depressed but equal. Fundi—retinal veins enlarged; both retinæ suffused but no edematous blurring limited to the optic disks. Lumbar puncture—straw-colored cerebrospinal fluid under a slightly increased pressure (12 mm.).

Last Report (May 20, 1918—18 months after injury).—Patient is working daily but is still using alcohol to excess. After being transferred to Bellevue Hospital, patient had remained on Blackwell's Island for a period of 3 months, so that his condition was good at discharge; while there, he received vigorous luetic treatment, including 7 intravenous injections of salvarsan, but since leaving the hospital the patient has had no further treatment. All efforts to examine the patient have been unsuccessful so that an accurate knowledge of his present condition is not possible.

Remarks.—It is rather unusual for a patient, whose resistance had been undoubtedly so lowered, both by chronic alcoholism and by lues, to have made such a comparatively excellent recovery following the cranial injury; patients of this type are most susceptible to brain injuries and their mortality is high in that acute cerebral edema and its resulting medullary compression and medullary edema occur most easily and to an extreme degree in this type of patient. The fracture of the skull, extending through the left middle ear and thereby permitting the escape of intracranial hemorrhage and cerebrospinal fluid, undoubtedly afforded this patient a greater chance of recovery—both of life and of his former health; the onset, however, of delirium tremens following so closely the cranial injury is usually a most dangerous factor. The general epileptiform convulsions were probably due to the cortical irritation of a cortical edema associated with chronic alcoholism and lues as the toxic factors and the cranial injury merely precipitated their occurrence. The comparatively excellent recovery of this patient is most unusual.

The accurate measurement of the pressure of the cerebrospinal fluid is well illustrated in this patient; in the acute condition of cerebral edema and intracranial hemorrhage, the pressure as registered by the spinal mercurial manometer was 16 mm., whereas at discharge and following a marked improvement of the patient's condition the pressure had been lessened to 12 mm. The lumbar puncture at the same time afforded an accurate means of determining the luetic status of this patient both by the Wassermann test and the cell count.

As a routine procedure, when cerebrospinal fluid is removed at lumbar puncture or if the lumbar puncture has been performed primarily to measure its pressure, a specimen of it is always sent to the laboratory for a Wassermann test and, in most cases, for a cell count which indicates more accurately the degree of activity of the process if lues is a factor; the higher the cell count, the more active is the process. The condition of this patient would have been very confusing if a careful laboratory examination had not been made, and it emphasizes the importance of careful laboratory tests, particularly of the cerebrospinal fluid, as a routine procedure. Then again, the presence of cerebrospinal lues may not have been a definite factor at all in the condition of this patient, although it is difficult to conceive that it did not tend to render the patient more susceptible to the condition of post-traumatic cerebral edema.

The persistent impairment of the hearing to a marked degree is rather unusual in these cases of traumatic rupture of the tympanic membrane due to a fracture of the adjacent temporal bone—unless the condition is complicated by lues; usually the hearing improves markedly within 12 to 18 months. The presence of lues in this patient may be a definite factor in preventing the recovery of the hearing; so frequently in luetic patients following trauma of any part of the body, the tissue reaction and luetic exudate are so great and of such large amount that a complete recovery of the function of these tissues is prevented, and this is particularly true of the healing of lacerations of the tympanic membrane in that the membrane becomes unusually thickened and retracted from the new tissue formation; luetic treatment, however, may cause an improvement of this condition by facilitating its absorption.

Case 93.—Acute severe brain injury associated with mild signs of intracranial pressure and complicated by cerebrospinal lues. No operation. Luetic treatment. Improved.

No. 488.—Georgiana. Forty-two years. White. Married. Housework. United States.

Consultation with Doctor L. H. Finch, Amsterdam, N. Y., October 20, 1916.

Family history negative.

Personal History.—One child of 18 years of age. During past 10 years, patient has had severe headaches which have become gradually worse; increasing periods of depression and melancholia.

Present Illness.—Seventy-two hours ago, while talking to her daughter, patient had difficulty in making herself intelligible—used the wrong words, and realized it, but could not find the proper words to express her thoughts; she left the room to enter the bathroom and as she stepped upon the tiled floor, the patient slipped, falling headlong against the bathtub; immediate loss of consciousness. Three hours after injury, a general convulsion occurred without any localizing signs; one hour later, another similar convulsion was observed, and one-half hour later the third general convulsion occurred. Six hours after injury, patient became semiconscious and remained in this stuporous condition for two days.

Examination at consultation (October 20, 1916—72 hours after injury). —Temperature, 99.8°; pulse, 76; respiration, 20; blood-pressure, 124. Semiconscious and can be easily aroused; complains of severe frontal headache. Laceration of scalp over the right eye; careful probing reveals no fracture of underlying frontal bone. No signs of bleeding from nose, mouth or ears; no mastoid ecchymoses. Pupils equal and react normally to light. Reflexes: patellar exaggerated—left greater than right; no ankle clonus but suggestive left Babinski which, however, is not constant; abdominal reflexes not elicited. Fundi—retinal veins enlarged; nasal margins of both optic disks and nasal half of right optic disk obscured by edema. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately

14 mm.); specimen of the cerebrospinal fluid sent to laboratory for Wassermann test and cell count.

Treatment.—Expectant palliative. Patient continued in this semiconscious condition for almost a week; the pathological report of the cerebrospinal fluid was now returned as 4 plus and a cell count of 38 per c.mm. The immediate administration of luetic treatment—mercurial inunctions, potassium iodide by mouth, mercurial salicylate injections and salvarsan intravenously caused an immediate improvement so that within ten days after their administration, the patient became entirely conscious, the headaches lessened and an uneventful recovery from the acute condition occurred; ophthalmoscopic examination on the eighteenth day after the treatment began was negative. Six months after the injury, the cerebrospinal fluid gave only a faintly positive Wassermann reaction, whereas the cell count was only 16 cells per c.mm.

Last Report (October 28, 1918—24 months after injury).—The patient is in better health than during the past 8 years. No headaches and no longer has periods of depression and melancholia. Physical examination negative. Wassermann test negative, both of blood and of cerebrospinal fluid, although the cell count of the latter was 12 per c.mm.

Remarks.—It is conceivable that this patient was not only in the stage of cerebrospinal lues at the time of the injury, but also in that period preceding the clinical onset of possibly paresis itself. The cranial injury merely caused an acute cerebral edema to be superimposed upon the cerebral edema already present due to the luetic toxemia producing the mild condition of "wet" brain; any cranial injury, no matter how slight, would have precipitated this acute condition of cerebral edema and thus simulating an acute intracranial lesion of traumatic origin. The lumbar puncture, however,—so essential not only for the measurement of the pressure of the cerebrospinal fluid but also for the pathological report in all of these intracranial conditions—indicated the proper treatment, and the excellent result obtained by the luetic treatment is most gratifying. It will be necessary, however, to wait for a period of at least 5 years and even longer to estimate accurately the ultimate result.

The greater activity of the reflexes upon the left side, and the suggestive left Babinski and the signs of a greater increased pressure over the right cerebral cortex as revealed ophthalmoscopically, indicated a greater cerebral edema immediately underlying the area of the direct head injury (right frontal region) and due most probably to an acute cerebral edema; its cortical irritation is revealed in the epileptiform seizures which may have been Jacksonian in character.

CHAPTER XI

POST-TRAUMATIC NEUROSES

FOLLOWING cranial injuries, the condition of neurosis—solely a functional impairment and in no way associated with an organic lesion or change of tissue—is of very common occurrence. Post-traumatic neurosis may be designated as being a post-traumatic neurasthenia (nerve-tire), psychasthenia (mind-tire), or in general terms "a nervous breakdown"; the term neurosis itself is in more general use and the fact that the condition appears so frequently following cranial and brain injuries makes it necessary to discuss it briefly in a separate chapter.

It must be stated that in many of these patients who develop an acute condition of nervous instability—the so-called neurosis—following cranial injuries of even trivial character, that a very large percentage of these patients were unstable emotionally for periods of months or years even before the accident, and that the injury itself merely precipitated and externalized the underlying neurotic condition; the recent psycho-analytical methods of examination frequently reveal the predisposing causes for the emotional tension—domestic unhappiness, business worries, a craving for sympathy, etc.,—so that an accident and slight injury to the body, and especially to the head of a patient who is already in this emotional condition, is sufficient in itself to be and to produce the so-called "nervous shock"—that is, the injury in itself was merely an immediate and contributing cause of the acute neurosis, which in many of these patients would have eventually occurred if the underlying causes of business, domestic or social worries had continued.

There is, however, another factor in the production of post-traumatic neurosis which has commonly been overlooked; it is the emotional influence upon the patient of a future lawsuit for damages as the result of the injury. If the accident was the alleged result of an employer's negligence or carelessness or the defendant's fault, then the emotional reaction of the patient (and plaintiff) is one of mingled anger and resentment—"hurt from no fault of my own"; if, however, there is added to this natural reaction, which is in itself only a temporary one of weeks possibly, the question of a lawsuit for damages against the offending party, then there appears immediately the complication of not only increased existing complaints but of many new complaints of all kinds; if the patient has had some headache and general soreness throughout the body, there will appear within several days and surely after a lawyer has been consulted regarding a lawsuit (if a lawyer—the so-called "ambulance chaser"—has not already secured the signature of the patient to an agreement for a lawsuit even while the patient is in bed at the hospital and within several hours after the injury and the admission)—there will then appear the added symptoms of extreme weakness, nausea, dizziness, restlessness and irritableness, inability to sleep, spells of suffocation and that vast chain of neurotic complaints. If a satisfactory settlement of the suit is not made within a period of several weeks and the trial does not occur until about one year and even two or three years later, the emotional condition of the patient can easily be understood: frequent consultations with the lawyer, medical examinations of the patient by the defendant's doctors, attempts to secure a satisfactory financial settlement, the finding of all the witnesses and their stories, the ordeal of a court trial, the date of the trial which may be postponed for months—it is thus easily imagined the upset condition of the patient's mind and his emotions! By the time the trial does actually occur, the patient is really in a highly neurotic condition—the emotions so sensitized that they react to the slightest suggestion, complaints of every character and degree are present, while the mind subconsciously exaggerates these symptoms to a remarkable degree; their "threshold of consciousness" has become so lowered to external stimuli as the result of the continued introspection and preparation for the trial which has been rehearsed to themselves daily for months, that there is no part of their body which does not "feel badly," and particularly is this true of the head; hysterical spells are not uncommon and even seizures simulating epileptiform convulsions of the minor (petit mal) and major (grand mal) types may occur in the extreme cases.

During the past seven years, I have had an excellent opportunity of studying conditions of post-traumatic neurosis; associated with several hospitals and with an active ambulance service as maintained especially by the Polyclinic Hospital, I have examined in hospitals, in one week, as many as twelve patients having acute cranial injuries with and without a fracture of the skull and with and without a severe intracranial lesion. In this manner, I have been in a position to see these patients from the very beginning of their illness, and by a system of examining these patients once every six months, or at least receiving a report of their condition, it has been possible for me to ascertain their condition up to the present time. Of the patients who died in the hospital from the immediate effects of the cranial injury, autopsies were performed in almost every case—either by the coroner's physicians or by ourselves (permission having been obtained from the nearest relatives of the patients). The patients who recovered with and without operation have been repeatedly examined since their hospital residence; lawsuits (if these occurred) and also the present condition of the patients have been carefully recorded. As the hospital doctor of these patients, I have had an opportunity to study their conditions intimately and the results have been interesting as well as surprising. These patients having cranial injuries with and without a fracture of the skull and whether conscious, semi-conscious or unconscious, in whom after their admission to the hospital there were found definite signs of an increased intracranial pressure as ascertained by the ophthalmoscopic examination of the fundi of the eyes and at lumbar puncture by means of the spinal mercurial manometer with and without the presence of blood in the cerebrospinal fluid—these were the patients having a distinct intracranial lesion due usually to a "wet" edematous condition of the brain or to a hemorrhage of varying degree associated or not with a cerebral laceration (which is comparatively of rare occurrence); the patients in whom the intracranial pressure was high, whether there was a fracture of the skull or not, were operated upon to relieve this increased intracranial pressure by the drainage of the cerebrospinal fluid and free blood and they formed about one-third of the patients, whereas the remaining two-thirds of the patients in whom the intracranial pressure was not increased or only mildly so, with or without a fracture of the skull, these patients were treated by the expectant palliative method of absolute quiet, ice-bag to the head, catharsis and a liquid diet until the acute signs of the injury disappeared, and in the majority of these latter patients in whom no blood was found in the cerebrospinal fluid, the signs of an increased pressure, when present, would fade away during the residence of the patient in the hospital—a period usually of one or two weeks, so that at discharge the patient could be considered organically well. In other patients, however, the signs of a mild increase of the intracranial pressure would persist as demonstrated ophthalmoscopically by an enlargement of the retinal veins and an edematous blurring of the nasal margins of the optic disks; this definitely increased intracranial pressure in these patients was the organic cause for their headache and all of these patients had headache, dizziness, at times nausea and even vomiting; unless this increased intracranial pressure is gradually lessened by the natural means of absorption (and it usually is within a period of three to six months), then all of these patients develop a neurotic condition superimposed upon this definite organic basis resulting from the injury; their symptoms are increased both in severity and in number, and especially if a lawsuit is contemplated and the trial is in the future; whether this nervous tension and emotional instability tend to elevate the general blood-pressure enough to prolong the intracranial condition cannot be ascertained, since the normal blood-pressure of these patients before the injury is unknown. In brief, however, it is these patients having a definite though mild increase of the intracranial pressure for whom the risk of the cranial operation of decompression and drainage would not be justified, and in whom this mildly increased intracranial pressure would ordinarily be absorbed by the natural means of absorption—it is these patients who do not recover their former good health and ability to work unless this increased intracranial pressure is eventually relieved; they remain "nervous" and emotionally unstable, and although usually improved following the trial and the end of their lawsuit, yet they are not so well as before the injury.

On the contrary, however, those patients having a cranial injury, with and without a fracture of the skull, and especially in the absence of blood in the cerebrospinal fluid and in whom there are no signs of an increased intracranial pressure, and yet they complain, not only for weeks but for months following the injury, and especially if a lawsuit is pending, when frequently the symptoms increase both in severity and in number—these are the patients in whom their neurotic condition has no real organic basis, but it is rather due to business, domestic or social worries and most frequently of all—a lawsuit; these are the patients having a true post-traumatic neurosis with no underlying organic basis, who always improve following the successful settlement of their lawsuit—in fact, in many of these patients the improvement is almost immediate, so that within a week the patients declare that they feel almost "well again." I have seen this occur over and over again: the headache practically disappears, no dizziness, no nausea, a sense of relief both mentally and physically, no longer easily fatigued—in fact, almost a new person or like their old selves; they are able to sleep, become less irritable and in every way much better, if not entirely well. If, however, the lawsuit is not satisfactorily settled—for instance, the case is appealed or the trial is postponed several months or even a year, then I have observed these patients to continue in their neurotic condition—even worse than before the first trial, and in three patients whose lawsuits were eventually settled satisfactorily to them, each one of them made an excellent recovery—and each within a period of six weeks. That is, in these patients having the uncomplicated condition of post-traumatic neurosis and naturally in the absence of a definite organic basis and especially of an increased intracranial pressure, these patients all improve after the successful termination of their lawsuit, and they all make excellent recoveries and regain their former good health, unless even before the cranial injury they were emotionally unstable—and some of these patients are so constitutionally. One of my patients who had been knocked down by a taxicab and had suffered only a slight cranial injury with a small laceration of the scalp—this patient developed a marked neurotic condition and chiefly because the offending chauffeur could not be located and punished; four months later when the guilty chauffeur was finally arrested, the patient's entire nervous condition immediately improved and the neurosis lessened daily until two weeks later when it was ascertained that the man was a bankrupt—then a relapse occurred and continued for a period of five months when it too gradually disappeared. The patients, however, who have the neurosis superimposed upon a definite organic lesion such as a mild increase of the intracranial pressure resulting from a traumatic cerebral edema of moderate degree—these patients also improve after a satisfactory settlement of their lawsuit but not to the extent of the former true neurotic patients; unless the increased intracranial pressure ultimately becomes absorbed, then these patients will continue to have complaints, especially headache of varying degree, change of disposition to either the depressed or irritable and restless type, early fatigue and the other symptoms as well as the definite signs of a mild intracranial lesion.

It is thus seen to be of the greatest importance, both to the patient and to the community, that these two conditions—a true post-traumatic neurosis and a definite organic lesion with a neurosis superimposed upon this organic basis, should be differentiated and they usually can be by careful and repeated neurologic examinations, and especially by competent ophthalmoscopic tests and the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer—the most accurate method now known for ascertaining the presence or not of a mild increase of the intracranial pressure. Only too frequently the medical examination of these patients is a cursory and superficial one—the diagnosis being considered either as one of no importance at all or at most a neurosis (and thus favorable to the defendant), or as being a very serious condition of mental and physical impairment, probably of permanent duration, and fraught with many dangers such as epilepsy, insanity and numerous others (and all favorable to the plaintiff). It is unfortunate and lamentable that these patients cannot be examined by a competent medical commission—the patient being placed in a hospital under observation for a period of at least one week, careful neurologic and psychiatric examinations impartially made and especially the registration of the intracranial pressure—it would then be possible for an impartial report of the real condition to be made and thus eminently fair to both the patient and the defendant—and to the community at large; under the present system of retained doctors, these patients and particularly the ones having this type of conditions are notoriously maltreated and misjudged—either intentionally or not. Wassermann examination of both the blood and cerebrospinal fluid and its cell count could and should also be made at the time of the lumbar puncture in order to exclude syphilis as a factor in the condition: it occurs only too frequently that a trivial blow upon the head of a patient in the latent stages of neuro-syphilis precipitates a more active process which now appears apparently for the first time and progresses rapidly into the various forms of cerebrospinal lues and even paresis. A careful urine examination to exclude the more serious types of nephritic disease, diabetes and the other toxic factors of a chronic edema of the brain which may have existed even before the cranial injury or at least made it possible for a chronic cerebral edema to persist; cardio-vascular diseases and especially arteriosclerosis are very influential predisposing factors in the causation and prolongation of this cerebral edema. On account of the above considerations, it is of the utmost importance that these patients should be examined very thoroughly in order to exclude all organic diseases in both the diagnosis and the prognosis—as well as to assure the appropriate treatment to the patient himself.

It cannot be doubted that many of these patients having post-traumatic neuroses consciously exaggerate their symptoms to a greater or less degree, and almost always, subconsciously at least, during the interval between the cranial injury and the trial for damages, or during a period of business or domestic worries—the cessation of which results in an immediate improvement of the condition of the patient; it is very easy and so natural for these nervous patients to ascribe any business failures and lack of capability to their condition of poor health—an excuse which merely permits the condition to become worse; also in domestic unhappiness, the craving for sympathy usually tends to increase the severity and the number of the complaints.

The more common signs frequently associated with conditions of post-traumatic neurosis are coarse irregular tremors and ataxia of the hands, a fine tremor of the eyelids upon closing the eyes and very active reflexes; no Babinski reflex, however, can be elicited in any of the true cases, although if the neurosis is superimposed upon an organic intracranial lesion, then it is possible for this characteristic dorsal flexion of the big toe upon plantar stroking to be present; it is a most important sign, when present, in the differential diagnosis—it cannot be simulated successfully. Conscious malingering and "faking" do occur, but it is usually so palpable and obvious to a competent and trustworthy medical examiner that mistakes in their diagnosis rarely occur.

In this connection it may not be irrelevant to mention again the comparative unimportance of the X-ray findings—unless the fracture is a depressed one of the vault of the skull and associated with a tear of the underlying dural covering of the brain. Linear fractures of the skull—vault or base—and not associated with a definite increase of the intracranial pressure are of no more significance in the treatment and the prognosis than that the original cranial injury was of sufficient force (and in many patients the blow need not be a powerful one) to cause a break in the continuity of the bone; a linear fracture of the skull in itself does not presuppose and rarely indicates a serious brain injury unless associated with a high intracranial pressure resulting from hemorrhage or cerebral edema; in fact, in many patients the fracture of the vault permits an intracranial hemorrhage and an excess of cerebrospinal fluid (cerebral edema) to escape through the line of fracture into the subcutaneous tissues of the scalp, or of the base into the openings of the nose and ears, and thereby lessens an increased intracranial pressure so that the danger of the cranial operation of decompression and drainage is avoided and the convalescence hastened, while the risk of infection through the line of fracture into the nose and ears has been slight and not a very probable one. The exhibition in court, therefore, of X-ray pictures and plates of fractures of the skull should be of significance only in establishing the fact that a cranial injury has occurred, but whether the cranial injury has really caused a serious injury to the brain—that can only be determined by the careful and thorough neurologic and psychiatric tests as outlined above.

Post-traumatic Neuroses

Case 94.—Cerebral concussion; recovery complicated by the condition of post-traumatic neurosis.

No. 474.—Elizabeth. 28 years. White. Married. Clerk. United States.
Admitted November 23, 1915. Polyclinic Hospital.
Discharged December 8, 1915—15 days after injury.
Family history negative.

Personal History.—Usual childhood diseases. Appendicectomy at 14 years of age. One child 10 years of age; 6 months before its birth, the left breast of the patient was removed on account of a "lump"—the size of a walnut; no recurrence.

Present Illness.—While in the lavatory of the factory in which she is employed, patient was struck by a marble slab of about "seven feet" in length, which had fallen from the ceiling upon her head; unconscious for several minutes and then brought by the ambulance to the hospital in a semiconscious condition.

Examination upon admission (40 minutes after injury).—Temperature, 98.2°; pulse, 88; respiration, 22; blood-pressure, 128. Well nourished and developed; appendix scar is noted and the left breast is absent. Conscious, but stuporous and drowsy. Complains of severe pain in head, especially upon right side over the temporo-parietal area. No laceration of the scalp. No bleeding from nose, mouth or ears; no mastoid ecchymoses. Pupils equal and react normally. Reflexes: active but equal; no Babinski; abdominal reflexes present and equal. Fundi negative. Lumbar puncture—cerebrospinal fluid perfectly clear and under normal pressure (approximately 8 mm.).

Treatment.—Expectant palliative.

Examination (12 hours after admission).—Temperature, 99.4°; pulse, 84; respiration, 20; blood-pressure, 134. Conscious; still complains of severe pain "throughout head." No cranial ecchymoses. Reflexes: patellar, active but equal; no Babinski; abdominal reflexes present and equal. Fundi —distinct congestion of veins of both retinæ with slight blurring of nasal margins of optic disks, especially in right fundus. X-ray (Doctor G. W. Welton)—"no evidence of fracture."

Examination (4 days after admission).—Temperature, 99°; pulse, 84, respiration, 22; blood-pressure, 136. Patient is conscious and is making an excellent recovery; headaches not so severe. Reflexes: patellar, active but equal; no Babinski. Fundi: slight dilatation of retinal veins; otherwise normal. Urine negative.

Examination at discharge (15 days after admission).—Temperature, 98.8°; pulse, 78; respiration, 20; blood-pressure, 132. No complaints except for a slight general headache and "soreness all over." Reflexes negative. Fundi negative.

Examination (May 10, 1916—5 months after injury).—Numerous complaints: "Headaches all of the time; dizzy spells and attacks of faintness so that cannot work as formerly; unable to sleep; no appetite; loss of weight." Patient has instituted suit against the owner of the factory in which she was injured; is being treated by various cults—osteopaths, herb doctors and Christian Scientists. Pupils equal and react normally. Reflexes—patellar, active but equal; no Babinski; abdominal reflexes present and equal. Fundi negative.

Examination (February 12, 1917—14 months after injury).—Same complaints as at the previous examination in May; headaches, however, are worse—"driving me crazy." Reflexes—active but equal; no Babinski. Fundi negative.

Last examination (January 14, 1918—25 months after injury and 2 months after the successful termination of her lawsuit from which she received $2800).—"Ever since Christmas (3 weeks before) I have felt much better; a slight headache in the morning occasionally; no dizzy or fainting spells; sleep fine; good appetite and gaining in weight." Reflexes active but normal. Fundi negative.

Remarks.—The almost immediate improvement occurring in this patient's condition upon the satisfactory settlement of her lawsuit for damages for her injury—this result is not unusual in patients having similar cranial injuries and complicated by a lawsuit for damages; if the patient should become well (and they rarely do) while waiting for the case to come to trial, the patient knows that the probability of receiving a large settlement or judgment will be lessened—and in the large majority of patients, it is "human nature" for them not to "get well." However, after a satisfactory settlement of the lawsuit, then the percentage of recovery within one year following the lawsuit is almost 100 per cent. Great care must be by the physician in each of these patients to make the most car thorough examinations of the patient in order to ascertain, without a question of a doubt, that there is no organic lesion present, before the statement should be made that the condition is merely a functional one—a post-traumatic neurosis. Each patient should be placed in a hospital for a period of one week, at least, so that careful and repeated neurological examinations can be made, especially the reflexes tested and any sensory impairment elicited—whether objective or subjective, daily ophthalmoscopic examinations and a measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer at least once and if there is any doubt, then the test should be repeated; a Wassermann test of the cerebrospinal fluid is also thus possible as well as a careful cell count. Very few mistakes will be made in considering true organic conditions as functional and *vice versa* if careful examinations are made over a period of one week as illustrated above.

The neurosis of this patient was unfortunately complicated by a lawsuit, but the condition itself was undoubtedly prolonged and rendered less susceptible to rational treatment by her being treated successively by osteopaths, chiropractics, herb doctors and last and least, Christian Scientists.

The underlying basis of the condition in many of these patients, if the lawsuit is excluded, is the fear that they will "go crazy" as the result of the head injury, because a friend possibly has been sent to an insane asylum years after a head injury; this fear having been dispelled from many of the patients' minds, an immediate and marked improvement frequently occurs. It can be easily imagined how the condition of these patients can be aggravated if, in addition to their fears of future mental derangement, there should be present a feeling of resentment toward the employer as being the cause for the injury and that the patient is entitled to a substantial financial recompense: the condition would become worse rather than stationary during the years following the injury and then a marked improvement would undoubtedly occur within a short time after a satisfactory settlement of the lawsuit.

Case 95.—Severe cerebral concussion; recovery complicated by the condition of post-traumatic neurosis.

No. 674.—Louise. Twenty-seven years. White. Married. Actress. U. S.

Admitted August 31, 1916, Polyclinic Hospital.

Discharged September 17, 1916—17 days after injury.

Family history negative.

Personal History.—Appendicectomy at 7 years of age. Several attacks of "gall-bladder colic" during the past 10 years; never jaundiced.

Present Illness.—While crossing the street, patient was struck by a motor truck; unconscious; brought to the hospital in the ambulance.

Examination upon admission (45 minutes after injury).—Temperature, 97.8°; pulse, 104; respiration, 28; blood-pressure, 116. Well-nourished and developed; unconscious and in severe shock. Multiple contusions of the body—right side of head, shoulders, neck and chest. No bleeding from nose, mouth or ears; no mastoid ecchymoses. Pupils slightly dilated but of normal light reaction. Reflexes: patellar—very active but equal; no Babinski; abdominal reflexes can be elicited with difficulty and are apparently equal. Fundi (Dr. J. A. Kearney)—"General brick-red of entire retinæ: veins are attenuated; margins and disks are indistinct and appear to be edematous—right is more marked." Urine negative.

Treatment.—Expectant palliative; rectal enemata of hot black coffee; external warmth and absolute quiet to combat the shock.

Examination (18 hours after admission).—Temperature, 99.6°; pulse, 94; respiration, 24; blood-pressure, 128. Much better in every way. Conscious; complains of severe pain in head, right shoulder and about the sternum. No mastoid nor orbital ecchymoses. Pupils negative. Reflexes—knee-jerks active but equal; no Babinski; abdominal reflexes present and equal. Fundi—slight dilatation of retinal veins and general retinal suffusion; no definite blurring of optic disk margins. Lumbar puncture—cerebrospinal fluid clear and under normal pressure (approximately 8 mm.). X-ray (Doctor A. J. Quimby)—"negative for fracture of vault or base."

Treatment.—Expectant palliative continued; liquid diet.

Examination (6 days after admission).—Temperature, 99.4°; pulse, 86; respiration, 24; blood-pressure, 136. Irritable and restless; complains of severe pain "all over," especially in head and chest upon deep inspiration. Reflexes—active but otherwise negative. Fundi—no distinct edema of the optic disk margins. X-ray of chest "negative for sternum and ribs."

Examination at discharge (17 days after injury).—Temperature, 99°; pulse, 82; respiration, 24; blood-pressure, 132. Complains of headache and pain about the sternum; spells of dizziness upon arising suddenly. Complains of the negligence of the police in not locating and arresting the chauffeur. Reflexes active but otherwise negative. Fundi negative.

Examination (June 20, 1917—10 months after injury).—Complains bitterly of headache, dizzy spells and then of convulsive seizures of several minutes to one hour—"sudden loss of consciousness," "shaking all over" and frequently involuntary urination, biting of tongue and "arching" of the back; she and her husband say that these spells occur about once a week and usually at night. Tremor of both hands. A hospital residence of 2 days and examinations at this time did not disclose any abnormality or signs of organic disease. Reflexes active; otherwise negative. Fundi negative. Lumbar puncture—clear cerebrospinal fluid under normal pressure (approximately 6 mm.); Wassermann test negative and cell count was 4 cells per c.mm. It is now learned that the lawsuit takes place in one month; the patient worries "all the time" about the inability of police to capture the offending chauffeur.

Examination (September 12, 1917—13 months after injury and 2 weeks after close of lawsuit in which the patient received $2200).—Patient complains of her pain in a general way but not so much and in such de formerly she would recite her complaints one after the other, but now necessary to question her regarding whether she has headache, pain chest, etc.. and she always says "Yes," or "It bothers me a good Reflexes active; otherwise negative. Fundi negative. No longer an of the hands.

Examination (April 12, 1918—20 months after injury and 6 months after her lawsuit).—Patient joyfully tells of her good health. "I haven't had a spell since the trial, although I feel light-headed sometimes." No complaints other than a sense of constriction at times about the chest upon exertion. "The guilty chauffeur has been arrested and I now feel happy." Reflexes active but equal; no Babinski. Fundi negative.

Last Examination (September 23, 1918—27 months after injury and 12 months after the lawsuit).—"Feel fine except my money is all gone—I played the ponies." Husband says there have been no spells nor other complaints; occasional cold in the head. Reflexes active but otherwise negative. Fundi negative.

Remarks.—The condition of this patient was excellent at the time of discharge from the hospital and during the following 3 months there were few if any complaints, but when the possibilities of a lawsuit for damages were recognized, then the patient's entire condition became worse, a definite tremor of both hands appeared, the reflexes became more active and the patient developed a highly "nervous" condition. I do not wish to state that this patient intentionally and consciously exaggerated her symptoms and signs, but when she realized that it would be to her material advantage to be sicker than she possibly might in reality be, then she did become more ill in that her symptoms increased and even her signs were exaggerated—but not to the extent of organic disease. As soon as the lawsuit was satisfactorily settled, the condition of this patient rapidly improved and up to the present date (January 30, 1919), this patient has not had a single convulsive seizure (undoubtedly the former "spells" were hysterical in character) and the patient is a well woman in every way. I do not think this patient was malingering, but I do feel that both her symptoms and signs were exaggerated subconsciously, due to her highly neurotic condition resulting from the worry incidental to a future lawsuit and the possibility of a financial settlement.

This patient would have been "excellent material" for the formation of an extreme type of post-traumatic neurosis, even if the complication of a lawsuit had been absent; her condition would have been aggravated in every way, both subjectively and objectively, and because the driver of the truck was not apprehended, the nervous tension of this patient was probably prolonged months more than if the offending chauffeur had been satisfactorily punished within several weeks after the injury; this patient would not have sympathized with herself as much as she did because she now considered herself a sort of "martyr" and as one unjustly punished. I feel sure that if the offending driver had been arrested early and punished, she would immediately have felt better than she had for months, especially in the absence of a lawsuit, which prolonged the condition. In like manner, patients having lawsuits for damages are all benefited and feel "compensated" after a satisfactory settlement of the lawsuit.

CASE 96.—Severe cerebral concussion; recovery complicated by the condition of post-traumatic neurosis.

No. 241.—Sadie. Twenty-five years. White. Single. Housework tria-Hungary.

Admitted January 10, 1914, Polyclinic Hospital. Referred by Doctor Arnold Sturmdorf.

Discharged January 31, 1914—21 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While the patient was walking upon the pavement in front of a new building which was being erected, she was struck upon the top of the head by a heavy wooden beam; she was knocked down but not rendered entirely unconscious; temporarily dazed and was led by a friend to the hospital.

Examination upon admission (20 minutes after injury).—Temperature, 98.8°; pulse, 86; respiration, 26; blood-pressure, 128. Well-developed and nourished; complains of severe pain on top of the head and in the neck. Very drowsy—falling asleep while the examination was being made. Hematoma over left vertex. Apparent weakness of the entire left side of face. No bleeding from nose, mouth or ears; no mastoid ecchymoses. Left pupil: possibly larger than right; reaction to light sluggish. Reflexes: all increased—right more than left; no Babinski, however, and the abdominal reflexes are present and equal. Fundi negative.

Treatment.—Expectant palliative; the patient was admitted to the hospital for careful observation.

Examination (10 hours after admission).—Temperature, 99.8°; pulse, 84; respiration, 24; blood-pressure, 132. Conscious, very irritable and restless; complains of terrific headache, "ringing in the ears" and "all stiff." No mastoid ecchymoses. Weakness of left side of face (peripheral in type). Pupils—slightly dilated but equal, and react normally. Reflexes: markedly exaggerated but apparently equal; no Babinski; abdominal reflexes present and equal. Fundi: retinal veins enlarged—left possibly greater than right; no definite blurring of nasal margins of optic disks. Lumbar puncture—cerebrospinal fluid clear and under normal pressure (approximately 8 mm.). X-ray (Doctor A. J. Quimby)—"negative."

Treatment.—Expectant palliative continued; ice helmet and effective catharsis apparently afford great relief to this patient.

Examination (11 days after admission).—Temperature, 99°; pulse, 78; respiration, 24; blood-pressure, 136. Still complains of headache though not so severe. Sleeps most of the time and very heavily. Weakness of the left side of the face not so marked (the frontalis muscle being also affected, this condition was the peripheral type of facial paralysis and due undoubtedly to an edematous constriction of the left facial nerve itself). Pupils equal and react normally. Reflexes: all exaggerated but no Babinski could be elicited; abdominal reflexes present and equal. Fundi—slight edematous blurring along the nasal margins, particularly of the left optic disk; retinal veins enlarged.

Examination at discharge (21 days after admission).—Temperature, 98.8°; pulse, 80; respiration, 24; blood-pressure, 130. Headache persists; complains of dizziness and "I feel shaky all over." Weakness of the left side of face can be elicited only by tests of facial movements—raising eyebr showing teeth, shutting the eyes tightly, etc., and it is only then ol that the left side of the face lags slightly. Reflexes: active and equal; no Babinski. Fundi—retinal veins slightly enlarged but no definite edema about the optic disks can be discerned.

Examination (May 16, 1914—5 months after injury).—Patient complains of headache, especially in the morning, sleepiness and easily fatigued. Admits she is worried about her lawsuit and says she knows, "I'll feel better after it's all over," and—I agree with her. Pupils equal and react normally. Reflexes: active but equal; no Babinski; abdominal reflexes present and equal. Fundi negative; retinal veins not enlarged. Former weakness of the left side of face cannot be elicited by any of the usual tests of facial movements. In order to ascertain accurately the presence or not of an increased intracranial pressure as a possible cause of her complaints, a lumbar puncture was performed: the cerebrospinal fluid was clear and under normal pressure (approximately 8 mm.).

Examination (July 15, 1914—7 months after injury).—Patient still complains bitterly of headache, sharp pain in the left eye radiating to the left ear, unable to work on account of drowsiness and early fatigue. Patient desires me to testify for her in court if necessary. No weakness of the face can be ascertained. Pupils—equal and react normally. Reflexes—very active but otherwise negative. Fundi—possibly a slight dilatation of retinal veins but otherwise negative.

Examination (November 14, 1914—11 months after injury and 3 weeks after a settlement of the case out of court—patient receiving $1600).—Patient greeted me in the clinic of the hospital by saying, "I feel like a new woman since we're all through with that mess." Patient seemed pleased with the settlement and expressed surprise that her headaches had practically disappeared, except "when I stay up late"; patient has not, however, done any housework since she received her money. Reflexes—active but equal and apparently negative. Fundi negative.

Examination (December 10, 1916—35 months after injury and 23 months after the satisfactory settlement of the lawsuit).—Happily married and no complaints except for a pelvic condition following the birth of a child. Reflexes—active but otherwise negative. Fundi negative. Referred to Gynæcological Clinic.

Last Examination (September 10, 1918—57 months after injury and 49 months after the satisfactory settlement of her lawsuit).—No complaints except the worries of her 3 children. Occasional headache but "not bad." Reflexes: active but equal; no Babinski; abdominal reflexes not obtained (abdominal wall very lax and corrugated from the frequent recent pregnancies). Fundi negative.

Remarks.—The left facial weakness persisting for one month after the injury was undoubtedly due to a temporary edema of the left facial nerve, as the result of the head injury or possibly to a direct trauma to the nerve itself after it had left the stylo-mastoid foramen and had entered the posterior portion of the left parotid gland and, as all 3 branches of the nerve were involved, the compression of the nerve must have occurred before it bifurcated in the parotid gland. The usual site of the lesion producing a peripheral facial paralysis following head injuries is in the aqueduct of

Fallopius, in the petrous portion of the temporal bone as the nerve winds about the tympanum; any severe injury to this part, particularly a fracture of the base of the skull passing through the middle fossa and into the petrous portion of the temporal bone, will either cause a complete tear of the facial nerve itself (which is rare) or an edema of varying degree of the nerve and thus causing its temporary compression on account of its situation in its narrow bony aqueduct and therefore a peripheral paralysis of more or less severity results; usually this edematous compression is of only temporary duration—as a rule, not longer than 6 weeks. If the facial paralysis is of the peripheral type (the forehead muscles being involved, which is a point of differential diagnosis from the facial paralysis of central and cortical origin, causing the muscles of only the lower two-thirds of the face to be paralyzed) and it persists for more than one year and is thus apparently a permanent facial paralysis, then the operation of anastomosis of one-half of the proximal end of the homolateral hypoglossal nerve to the entire distal end of the impaired facial nerve must be considered, in order to cause a return of motor function to the paralyzed muscles supplied by the facial nerve. (Vide *J. A. M. A.*, May 11, 1918, p. 1354.)

The vast majority of these patients are not conscious malingerers and they would be diagnosed as having conditions of post-traumatic neurosis, whether associated with a lawsuit or not. The effect of the lawsuit, however, upon a patient is to change his or her attitude toward the complaints in that they are subconsciously exaggerated, their mind is increasingly directed and centered upon the complaints more and more as the trial approaches, a yearning for sympathy appears so that by the time the trial is started, the patient has reached a condition of nervous tension which may simulate a functional condition of almost any character, and if careful neurological examinations are not made it would be easy to confuse the condition with a definite organic disease. The trial over, and especially if successful from the patient's standpoint, then the relief of the nervous tension is great indeed and within several weeks, at most, the patient is enjoying excellent health! Such is the history of the large majority of patients having a true condition of so-called "post-traumatic neurosis." It is self-evident that the best treatment for these patients is a satisfactory settlement of the lawsuit, and no amount of medicine or psychotherapy will be of any marked and permanent benefit, unless the lawsuit be either satisfactorily settled or discontinued; I do not think it advisable to treat such patients medically for their neurosis while their lawsuit is still in the future—in the majority of patients all medical effort will be futile, and it is only after the lawsuit that a marked improvement is possible and—a satisfactory financial settlement is the panacea.

CASE 97.—Cerebral concussion; recovery complicated by the condition of post-traumatic neurosis.

No. 1045.—Harry. Thirty-six years. White. Single. Iron-worker. Sweden.

Admitted November 26, 1918—22 months after cranial injury, Audubon Hospital. Referred by Doctor W. H. Oliver.

Discharged December 14, 1918—18 days after admission.

Family history negative.

Personal History.—Always well and strong; of good habits.

Present Illness.—Twenty-two months ago (January, 1917), while patient was working in a factory, he was struck upon the head by a large wooden beam; immediate loss of consciousness for several minutes; no bleeding from nose, mouth or ears; taken to a hospital where he was discharged at the end of 10 days as "well." While in the hospital there were no signs of an increased intracranial pressure observed; the scalp had not been lacerated nor did there appear any orbital or mastoid ecchymoses. Patient complained, however, of constant dull headache, dizziness and weakness of the right side of body; also numbness of both hands and feet. During the past 22 months, patient has been unable to work owing to these subjective complaints, and although he has been repeatedly examined no definite lesion has been ascertained. He is receiving regularly his workman's compensation.

Examination upon admission (22 months after injury).—Temperature, 98.6°; pulse, 78; respiration, 18; blood-pressure 140. Well-developed and nourished; looks worried and anxious. No external evidence of the former head injury. Patient limps slightly upon the right leg and complains of severe headache throughout the examination. Hearing negative; otoscopic examination negative. No paralyses nor impairments of sensation elicited. Pupils equal and react normally. Reflexes—patellar active, right possibly greater than left (at a later examination this could not be confirmed); no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi—retinal veins of normal size; no edematous blurring of margins of optic disks. Lumbar puncture—clear cerebrospinal fluid and not under increased pressure (10 mm. as measured by the spinal mercurial manometer); Wassermann test negative and the cell count was 5 cells per c.mm. X-ray (Doctor A. J. Quimby)—"no fracture of the skull shown." Wassermann test of blood negative. Urine examination negative. The subjective weakness of the right hand and right leg are consciously exaggerated by the patient and all of the tests as to its real presence were negative; the patient insisted that he could not stand with his feet together and his eyes shut, and upon being tested for it he was unable to do so, but later in the examination when his mind was centered upon another test he was able to stand with his feet together and his eyes shut,—and perfectly. There is no nystagmus nor intention tremor, nor can any of the signs, including speech, of cerebellar disease be elicited; there is, however, some corneal and pharyngeal hypesthesia of a hysterical character. During the patient's residence in the hospital of 18 days, the above tests were repeatedly confirmed.

Diagnosis.—This condition is undoubtedly a functional one having both the elements of a hysterical character and of a post-traumatic neurosis complicated by the circumstances of his weekly compensation.

Prognosis.—This is the type of patient—and there are many of them following head injuries of varying severity and to whom weekly or monthly compensation is given,—who will not recover so long as compensation (no matter how small) is to be obtained by these patients having complaints chiefly of a subjective character and of a greatly exaggerated objective character, either in its entirety or partially. Under the present circumstances, this patient will indefinitely remain in the same condition, neither improving markedly nor becoming much worse—although the latter course may occur to some extent.

Treatment.—As long as the patient receives compensation, he will not become well—no matter what treatment is administered, medical or surgical. As a matter of treatment alone, if a final settlement financially could be made with the patient, then an early improvement will occur—sometimes almost immediately, so that the patient would be able to work within a period of 6 months or even earlier. There is no indication of any organic disease intracranially and no operation would be of any benefit (even psychically) to this type of patient.

Remarks.—Patients of this character are most difficult ones to treat in that the patient well knows that if he improves at all so that he is able to do light work, then his weekly or monthly compensation is diminished and at times, even abolished entirely. Besides, there is a marked tendency, if only subconsciously, for the patient under these circumstances of weekly or monthly compensation, to exaggerate his condition both subjectively and objectively—the greater the impairment, the larger the amount of compensation. It is easily seen that to attempt to treat medically such a patient successfully, the question of compensation should, if possible, be settled so that it does not complicate the successful treatment of the patient; in some of these patients a satisfactory financial settlement is the best medicine. Great care, however, should always be exercised by the physician in making most careful and thorough examinations in all of these patients before reaching the diagnosis of a functional condition.

CHAPTER XII

CHRONIC BRAIN INJURIES

IF depressed fractures of the vault are excluded, then chronic brain injuries are in no way dependent upon the question as to whether the skull has been fractured or not at the time of the cranial injury; just as in acute brain injuries, the presence or not of a linear fracture of the base of the skull is of little importance in estimating the true intracranial condition, the appropriate treatment and the prognosis, so in chronic brain injuries it is of little value to ascertain that a linear fracture of the skull has occurred at the time of the original cranial injury except as an indication that the injury was of sufficient force to cause a fracture of the skull; as is well known, however, in many patients following a cranial injury, the skull may not be fractured and yet the intracranial and cerebral lesion is frequently most severe and dangerous, both to the immediate life of the patient and to the future normality. Naturally, cranial röntgenograms should be taken in all of these patients for the purpose chiefly of excluding a depressed fracture of the skull; but linear fractures of the vault or of the base of the skull are of no diagnostic significance of the intracranial lesion—if one is present. The use of positive cranial röntgenograms in court as evidence of a permanent brain injury in these patients is more the result of enthusiastic ignorance than a real conception of the comparative unimportance of the linear fractures themselves.

In order to obtain more accurate data regarding the frequency of chronic brain injuries, I examined, in 1912, the records of three large hospitals in New York City of their patients having had acute brain injuries during the decade of 1900–1910. The average mortality from the acute brain injury was 50 per cent.; of the patients who survived following operation or no operation and were discharged as "well" or "cured," I could only locate 34 per cent. of them in 1912 on account of death from intercurrent disease, change of residence, and thus "lost," etc.; of these 34 per cent. of recoveries, however, I found that 67 per cent. of them had not been well since the head injury—"never the same man again," "always complaining," "cannot do a day's work," "queer ever since," "a bum," "a loafer" and the like; such were some of the minor complaints of both the former patient and the relatives—the latter observing the changes of personality following the cranial injury in a large number of the patients; the complaints of "severe pain in the head," "dizzy spells" and very infrequently but still an occasional patient "having convulsions"—this was indeed an impressive array of symptoms and also signs in two-thirds of the patients found, in many of whom a careful neurologic and ophthalmoscopic examination disclosed the definite signs of a persisting intracranial lesion. (At the time of these examinations in 1912, the spinal mercurial manometer was not in use and the importance of an accurate registration of the pressure of the cerebrospinal fluid in patients of this character was not fully appreciated by the medical profession.) Among these post-traumatic and chronic patients, there was a number of post-traumatic neuroses either of the simple type associated with business, financial and domestic worries, or of the complicated type superimposed upon a definite organic intracranial lesion and usually a chronic cerebral edema of mild degree; other patients exhibited increased and unequal reflexes, an occasional Babinski reflex, impairments of the special senses and the signs of an increased intracranial pressure as disclosed by the ophthalmoscopic examination of the fundi—usually an edematous blurring of the nasal margins and even the temporal margins of the optic disks, and in the absence of cardio-renal and cardio-vascular disease; the factor of chronic alcoholism so common in many of these patients was excluded as much as possible.

The usual intracranial lesion was apparently a chronic "wet" edematous condition of the brain following the cranial injury and due either to the residue of a supracortical film of hemorrhage which had not been entirely absorbed and thus blocking in greater or less degree the stomata of exit of the normal excretion of the cerebrospinal fluid into the cortical veins and sinuses, or to the continued presence of the acute cerebral edema immediately following the cranial injury but in milder degree owing to its partial but not complete absorption due to complications in the expectant method of treatment, such as alcoholism, intestinal and renal toxemia, mental and emotional strain and other harmful factors in the complete recovery of the patient. The presence of supracortical adhesions resulting from the former subdural hemorrhage was also a factor, and especially in the presence of an increased intracranial pressure with which they were usually associated. These findings were ascertained in a number of the patients at operation even at this late date following the acute intracranial injury and the results have been very beneficial in many of them. Naturally, cerebral lacerations and intracranial lesions destructive of brain tissue cannot be remedied and the patients are never operated upon unless associated with a definite increase of the intracranial pressure, which should be relieved and thus a chance for improvement is even possible in these patients by lessening the pressure upon the normal brain cells adjacent to the ones primarily destroyed; not only can the signs of impairment be improved but the symptoms of headache, dizziness, etc., be relieved and even entirely removed.

In brief, if depressed fractures of the vault (which should always be elevated or removed) are excluded, only those patients having chronic brain injuries associated with an increased intracranial pressure should be given the benefit of a subtemporal decompression in the hope and belief that a lessening of the increased intracranial pressure will permit a definite and permanent improvement, whereas those patients in whom there is no increase of the intracranial pressure are naturally not operated upon—no matter how extensive the mental or physical impairment is—since the damage in these patients was a primary one occurring at or due to the original brain injury, and the operation of cranial decompression, if indicated at any time, was then rather than months or years later, and especially now in the absence of an increased intracranial pressure. This view cannot be too strongly emphasized because operations are being advised in these latter patients with brain injuries in the absence of an increased intracranial pressure and the results are bad, and they cannot but be bad, since the intracranial pathology cannot now be remedied.

Traumatic Epilepsy.—The condition of post-traumatic epilepsy is a most discouraging one from an operative standpoint, in that it is usually the result of a condition which could have been relieved at the time of the primary cranial injury and thus the epileptiform convulsions could have been avoided. Naturally, depressed fractures of the vault should then be elevated or removed, for if permitted to remain until epilepsy of either the localized Jacksonian type or of the general convulsive type occurs (and it will occur in a large percentage of these patients), then it is frequently too late to obtain a good result even if the depressed area of bone or foreign body irritating the cerebral cortex is removed, and especially after the so-called epileptic "habit" (resulting from the chronic cortical irritability) has been established; a cranial operation at this late date will in many patients be followed by merely a temporary cessation of the "spells," and within a period of one to three years the convulsive seizures are as numerous if not more frequent than before the operation. In my opinion, a cranial operation is only indicated for those patients in whom the mental and emotional deterioration is slight, the epileptiform attacks few and of infrequent number, and in whom there is disclosed a marked increase of the intracranial pressure which is not secondary and due to the convulsions themselves; this latter differentiation can be ascertained by saturating the patient with triple bromides, luminal, etc., so that a convulsive seizure does not occur for a period of six weeks and then at the end of this non-convulsive period by estimating and comparing the intracranial pressure accurately by means of the ophthalmoscope and the spinal mercurial manometer with the intracranial pressure as registered before this non-convulsive period. In this manner, the increased intracranial pressure, if secondary, is excluded by its return to normal, whereas an increased intracranial pressure which is primary to the convulsions can be thus ascertained definitely and by an operative removal of the original irritative focus, as in depressed fractures of the vault, and a lowering of the increased intracranial pressure in the other patients and thus a lessening of the cortical cerebral irritation, the patient is given in these selected cases a definite chance of a permanent improvement—if not, in rare cases, a cure itself. This careful selection of patients, both as to their general condition of mental and emotional deterioration, the infrequency of the convulsive seizures and the presence of a marked increase of intracranial pressure which is not secondary to the convulsions themselves (whether there is a depressed fracture of the skull or not)—these are the comparatively few patients and the only ones who can be benefited by any cranial operation of either an elevation or removal of the depressed area of bone or other foreign body or by means of the operation of cranial decompression alone. This is, however, late treatment of these patients—the condition should have been avoided and prevented (and it usually can be at the time of the acute cranial injury); many of them become derelicts so that any treatment, operative or not, cannot make it possible for them to regain their former good health and normality, but in the selected patients as outlined above, it is not only justifiable but the only method now known of affording these patients a chance of recovery.

A. CHRONIC BRAIN INJURIES ASSOCIATED WITH A DEPRESSED FRACTURE OF THE VAULT.

In the presence of an old depressed fracture of the vault, which should have been elevated or removed at the time of the cranial injury if it had not been overlooked and not diagnosed, or (and it unfortunately happens very frequently—even in these more recent days) in the belief that a depressed fracture of the vault would not cause future impairment, because "John Jones had a similar depressed fracture and it did not cause trouble in his case" and a similar method of limited reasoning,—it is in these patients that merely an elevation or more usually a removal of the depressed area of bone, in the absence of an increased intracranial pressure, is alone sufficient to obtain a marked improvement in many of the patients—months and even years after the original cranial injury; in other patients, however, little or no improvement results; the patient, however, should be given this chance of improvement. If necessary, the underlying dura may be incised with safety in the absence of a marked increase of the intracranial pressure and the supracortical or cortical cyst, with and without the presence of adhesions resulting from the former hemorrhage, may be incised, and the outer wall removed.

In the presence of a definite increase of the intracranial pressure associated with the depressed fracture of the vault, then it is better surgical judgment to perform a subtemporal decompression first, so that the pressure is thus safely lowered and then the depressed area of the vault can be elevated or removed, and yet little or no danger to the underlying more highly developed cerebral cortex has occurred; in those patients in whom the site of the depressed bone is overlying the large sinuses of the occipital area, it is frequently wiser for fear of the complications of hemorrhage not to attempt the local removal of the depressed bone, but to depend upon the general lessening of the intracranial pressure by means of the subtemporal decompression alone; later if considered necessary, then the depressed area of bone may be elevated or removed.

A. *Old brain injuries associated with depressed fractures of the vault. No operation. Symptoms and signs persisting. Operation advisable; consent not obtained.*

CASE 98.—Old brain injury associated with a depressed fracture of the left occipital bone; right homonymous hemianopsia. No operation. Symptoms and signs persisting. Operation advisable but refused.

No. 161.—Clarence. Twenty-four years. White. Single. Iron-worker. United States.

Admitted July 20, 1914—3 months after injury, Hospital for the Ruptured and Crippled. Referred by Doctor W. L. Sneed.

Discharged August 2, 1914. Operation refused.

Family history negative.

Personal History.—Always well and strong. Three months ago (Ap 1914) while at work, patient fell a distance of 35 feet from a plank, st upon the back of his head; immediate loss of consciousness; taken to a hospital, where the laceration over the left occipital area was sutured, and although it was noted that there was a depressed fracture of the left occipital bone, no attempt was made to elevate or remove it. No bleeding from the nose, mouth or ears. Patient was discharged from the hospital 25 days after admission as "well." He was, however, very confused mentally and did not remember his hospital residence or discharge; since then he has had severe frontal headaches with dizziness, emotional instability and impairment of vision.

Present Illness.—One day ago, patient came to hospital complaining of acute pain at the site of former laceration and upon examination a swelling, the size of an English walnut, was found at the site of former injury; it was incised, allowing creamy pus to escape (bacteriological report (Doctor Jeffries)—"staphylococci"); it was apparently merely an infected hematoma at the site of former laceration and the patient was immediately relieved of the local pain and tenderness. Patient, however, still complained of a general headache—particularly in the frontal region, dizzy spells, inability to "concentrate my mind," impairment of memory especially for recent events, and inability to see objects in the right half of the visual field. Brother says he is a "changed boy"; very irritable, so much so that they "fear to cross him"; "he seems in a trance at times."

Examination (3 months after injury—consultation with Doctor Sneed). —Temperature, 98.8°; pulse, 82; respiration, 18; blood-pressure, 128. Laceration of left occipital area bandaged; just posterior to the left mastoid process is a small purulent sinus. Pupils—left contracted and reacts to light sluggishly; consensual light reaction present. Reflexes—patellar very active, right greater than left; no ankle clonus but tendency to a right Babinski; abdominal reflexes—right depressed. Fundi—retinal veins dilated; nasal margins of both optic disks, and especially the left, blurred by edema; left physiological cup shallow from new tissue formation. Visual fields—complete right homonymous hemianopsia, otherwise fields of vision are normal (Fig. 124). No nystagmus; ocular movements normal. Lumbar puncture—clear cerebrospinal fluid under increased intracranial pressure (approximately 14 mm.); Wassermann reaction negative and cell count was 6 cells per c.mm. X-ray (Doctor B. C. Darling).—"depressed fracture of left occipital bone with linear fracture extending downward toward the foramen magnum."

Treatment.—The local infection at the site of the laceration and depressed fracture was treated by free drainage and then expectantly in the hope that the infection would disappear; then if the signs of the local lesion of the left occipital lobe—especially the right homonymous hemianopsia, did not improve, it would be advisable to perform, first, a left subtemporal decompression to lessen the general increased intracranial pressure, and then a local exploratory procedure over the left occipital lobe in the hope that the lesion was one of compression rather than a primary destruction of the occipital cortex itself. After the local infective process subsided, however, the patient refused to have any further operative treatment, and the patient was therefore discharged in the same physical condition as upon his entrance with the exception of the disappearance of the local infective process.

Examination (September 18, 1915—17 months after injury).—Patient still complains of headache, although possibly less severe than formerly; unable to work at his former trade upon bridges for fear of falling in a "dizzy spell." Relatives state that the patient is not quite so irritable as he was during the first 6 months following the injury; impairment of the right half of the field of vision persists. Laceration over the left occipital area entirely healed; no tenderness. Pupils unequal, left smaller than right, and the reaction to light is sluggish. Reflexes—patellar very active, right more than left; tendency to right Babinski persists; abdominal reflexes—right obtained with difficulty. Fundi—retinal veins enlarged; nasal margins of both optic disks still blurred.

Treatment.—Even at this late date, the patient was advised to undergo, first, a left subtemporal decompression and then a local exploratory operation with an elevation or removal of the depressed left occipital bone. The patient refused to have any operation performed. (Patient is receiving compensation each week and says: "If I die from an operation, I lose out, and if I get well I also lose out by losing my compensation.")

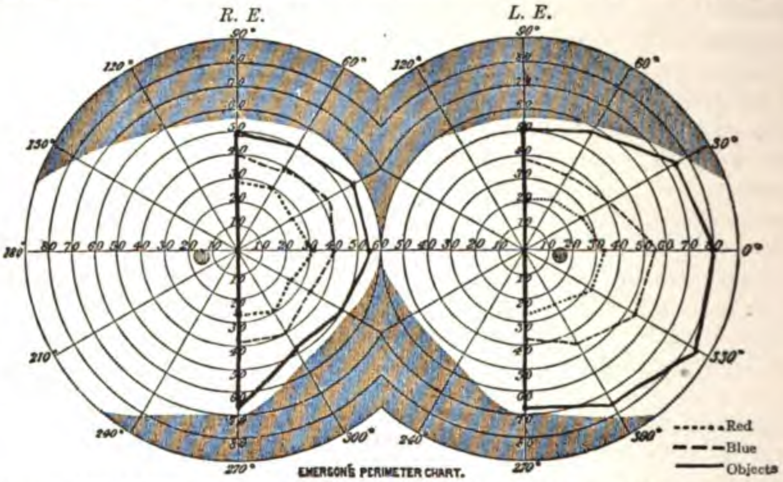

FIG. 124.—Total right homonymous hemianopsia following a depressed fracture of the left occipital bone. Normal consensual pupillary light reflex is present.

Last Report (May 18, 1918—49 months after injury).—Brother states that the patient is in practically the same condition—no better and no worse; less irritable than formerly but, on the contrary, he is becoming less and less interested "in things"—seldom goes out and will no longer work. Brother is trying to arrange for patient's admission to the hospital.

Remarks.—It is unfortunate that an early elevation, or better, a removal of the depressed area of the left occipital bone was not performed, and if a subdural hemorrhage was present, then its early drainage; naturally, if there were signs of an increased intracranial pressure, then a left subtemporal decompression would have been advisable first, to be followed then by the local operation.

The right homonymous hemianopsia may have been due to a laceration of the left occipital cortex itself and therefore an entire recovery of the visual field could not be expected following an operation, and yet the presence of an increased intracranial pressure would encourage us to believe that even though the cortex itself was lacerated, yet there would be some contiguous cortical cells not primarily destroyed by the laceration but merely compressed and functionally impaired due to the resulting increased intracranial pressure. For this reason, in the presence of an increased intracranial pressure, the condition should not be considered hopeless—the hope being that the left occipital cortex was being compressed by the depressed area of bone or by an extradural or a subdural clot; the presence of the consensual light reaction and its being normal, places the lesion behind the optic chiasm. At first, the condition was considered as possibly due to an abscess formation underlying the site of fracture, but the fact that the purulent discharge disappeared within a few days following appropriate treatment and that no untoward signs appeared within the following 2 years would indicate that the infective process had been limited to the scalp alone.

It is to be regretted that this patient was not operated upon earlier when advised, as it would seem from the last reports that the patient was deteriorating both mentally and physically, undoubtedly due to the local cerebral lesion plus the increased intracranial pressure.

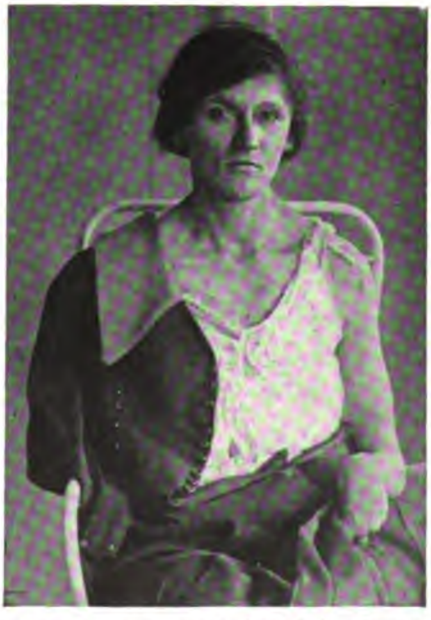

Fig. 125.—Left spastic hemiplegia affecting chiefly the left arm in a patient as the result of two bullet injuries of the brain. The flexor contractures of the left arm at the elbow and wrist are shown.

Case 99.—Old severe brain injury associated with a penetrating bullet injury of the brain and with a mild increase of the intracranial pressure; slight left hemiplegia. No operation; symptoms and signs persisting. Operation advisable.

No. 287.—Ella. Twenty-one years. White. Single. Home. U. S.

Admitted June 5, 1914—5 years after injury. Hospital for the Ruptured and Crippled. Referred by Doctor B. H. Whitbeck.

Discharged June 20, 1914—15 days after admission.

Family history negative.

Personal History.—Always well and strong until cranial injury. Five years ago (when patient was 16 years of age), she was accidentally shot twice with a .32-calibre revolver bullet through the right frontal bone near the midline and also at the side—the latter bullet going obliquely downward so that it lodged posteriorly and below the left orbit, whereas the first bullet remained in the right frontal lobe. Immediate loss of consciousness; no bleeding from nose, mouth or ears. Patient was taken to a hospital, where she remained 4 weeks; the wounds of entrance over the right vertex were carefully probed, "cleaned" and drained; naturally, no attempt was made to remove the bullets. Patient regained consciousness after five days and gradually improved so that, when she was discharged from the hospital, she complained only of severe headache and the weakness of the left side of the body. During the past 5 years, she has complained of continuous daily headache, but the weakness of the left side of the body has improved so that now the left arm is chiefly impaired. Patient has had competent medical treatment, massage, exercises, etc.

Examination upon admission (5 years after injury).—Temperature, 98.6°; pulse, 82; respiration, 22; blood-pressure, 128. Well-developed and nourished. No definite mental impairment but slight emotional instability—irritable and unable to control "her temper" under slight provocation. Over the right half of the frontal bone are the two scars of bullet entrance in the scalp. Mild left spastic hemiplegia, more marked in left arm and least in the left side of face; severe flexor contracture of left arm at elbow and wrist (Fig. 125); no sensory impairment can be elicited. Pupils—right slightly larger than left but reaction to light is normal. Reflexes—patellar very active, left more than right; exhaustible left ankle clonus and suggestive left Babinski; deep reflexes of left arm exaggerated; abdominal reflexes present, left less active than right. Fundi—retinal veins enlarged; nasal margins of both optic disks and nasal half of right optic disk blurred by edema. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 14 mm.); Wassermann test negative. X-ray report—"two irregular bone defects of 1 cm. in diameter of the right half of the frontal bone; two bullets are clearly shown; small bony spicules are visible along the course of the bullets and also possibly particles of lead itself" (Figs. 126 and 127).

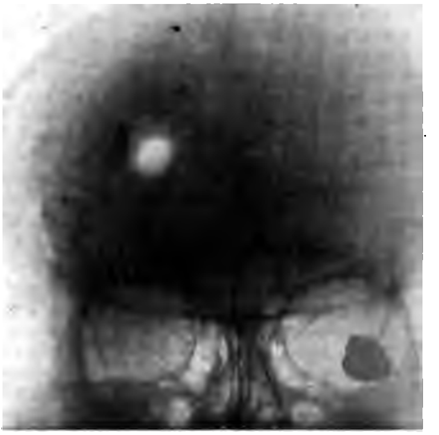

FIG. 126.—Antero-posterior view showing the two bullet defects of entrance in the right frontal bone and the two bullets themselves—one in the right frontal lobe and the other one just posterior to the left orbit. Bony and leaden particles can be observed in the paths of the bullets.

Treatment.—The presence of the definite increase of the intracranial pressure due most probably to a chronic cerebral edema following the cerebral trauma and the intracranial hemorrhage at the time of the injury, and since competent medical treatment has not caused a complete lessening of the increased intracranial pressure, a right subtemporal decompression was considered advisable to cause a cessation of the headache, possibly a greater ultimate improvement of the left hemiplegia and, by diminishing the cortical irritability, to lessen the great danger of later epileptiform seizures—a most serious complication. The parents of the patient felt that an operation was "not advisable" at this time and consent was therefore not obtained.

Examination (May 5, 1916—7 years after injury).—Patient is in practically the same condition as at the preceding examination; the headaches occur daily in varying severity, while the mild left hemiplegia, particularly of the arm, still persists. Patient wishes to postpone operation until a later date so that "I shall be stronger."

Last Report (September 10, 1918—9 years after injury).—Sister states that the patient is in the same condition as before but "is afraid of an operation."

Remarks.—It is inconceivable that a bullet could penetrate the brain as in this patient, and yet there not form a large amount of subdural hemorrhage and cerebral edema; for this reason, it is always advisable in the acute cases of bullet injury of the brain when the bullet has penetrated the dura and the cerebral tissue itself, to perform an ipsolateral decompression and drainage of this intracranial hemorrhage and cerebral edema, and thus lessen the great danger of future complications, especially convulsive seizures, severe headache, etc. Naturally, it is meddlesome surgery to attempt extraction of bullets and small foreign bodies which have penetrated the brain subcortically—the risk of causing greater cerebral damage is far more than the presence of the bullet itself. If the decompression is performed, and, if necessary, a bilateral decompression and drainage, then the intracranial pressure will be lowered, the hemorrhage drained and thus the patient be given the best chance of the greatest ultimate improvement. Cerebral tissues destroyed by the passing of the bullet naturally do not regenerate, but the adjacent fibres and cells are merely compressed by edema or hemorrhage and a relief of this pressure by the subtemporal decompression and drainage will afford the earliest and greatest immediate and ultimate improvement, and also lessen the danger of future complications.

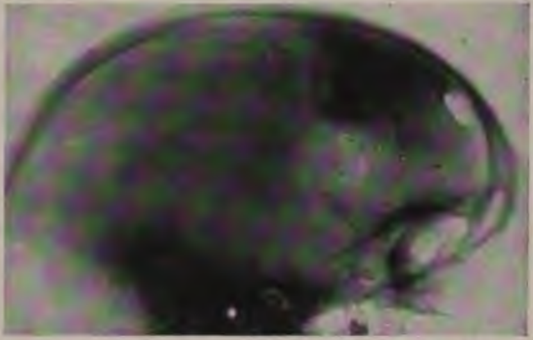

FIG. 127.—Right lateral view of the same patient showing the two bullets and the bony defects of the right frontal bone as the result of their entrance.

CASE 100.—Old severe brain injury associated with a depressed fracture of the vault and marked signs of an increased intracranial pressure; immediate removal of the depressed area of bone. Symptoms and signs persisting. Decompression operation advisable.

No. 1029.—Donald. Twenty-seven years. White. Single. Brakeman. England.

Admitted September 10, 1918—9 months after injury. Polyclinic Hospital. Referred by Doctor R. H. Dennett.

Discharged September 18, 1918—8 days after admission. No operation.

Family history negative.

Personal History.—Always well and strong until the following cranial injury: 9 months ago (December 13, 1917), while at work, patient was caught between two freight cars and an iron bolt forced through the bone of the left occipital area; no loss of consciousness; no bleeding from nose, mouth or ears. Patient now remembers that he could see a little out of the left eye for several hours after the injury and then a gradual complete loss of vision of the left eye and of the temporal half of the field of vision of the right eye. Eight hours after the injury, the patient was operated upon at a hospital in Schenectady and the depressed area of bone in the left occipital region was removed; the patient made an excellent operative recovery but no improvement of the vision of the left eye or of the temporal half of the field of vision of the right eye has occurred. During the past 8 months since the injury, patient has complained of persistent frontal headache, spells of "light-headedness"—at times of sufficient severity to cause him to stagger, but he has not fallen to the ground; tires very easily so that he has been unable to work throughout one whole day; has become very irritable, especially over trifles, and is so restless that "I can't remain in one place or do one thing longer than several minutes at a time; I must then get up and go." Patient now comes to the hospital in the hope that something can be done for him so that he can "do a full day's work." No convulsive seizures, but he has had a momentary loss of consciousness several times, and especially during meal-time.

Examination upon admission (9 months after the injury).—Temperature, 98.6°; pulse, 70; respiration, 18; blood-pressure, 138. Unusually well developed and nourished (Fig. 128). No mental retardation apparent but a definite emotional instability—very irritable, resents questioning and is very restless—"I must always be moving and doing something." Cranial examination reveals a bony defect in the lower posterior portion of the left parietal bone—the size of 3 cm. in diameter; it is not depressed and pulsation is palpable. (Apparently an osteoplastic flap operation had been performed at the time of the injury and the depressed area of the vault had been removed; it is not known whether the dura was opened or not.) No paralyses nor sensory impairments. No impairment of hearing; otoscopic examination of both tympanic membranes negative. Complete loss of vision of left eye, although patient can distinguish light but no objects; loss of vision of the temporal half of field of vision of the right eye; that is, a temporal hemianopsia of right eye (Fig. 129). Pupils—equal and react n
mally, left pupil possibly slightly larger than right and its reaction to
not so active (this variation, however, is not constant at several exa
tions); consensual light reaction present and normal. Reflexes—pa
exaggerated, right more than left; right exhaustible ankle clonus and
gestive right Babinski; right abdominal reflex less active than left. F
retinal veins slightly enlarged; indistinct hazy edema about the na

gins of both optic disks. (Left optic disk appears smaller than normal, but no signs of atrophy, either primary or secondary.) Lumbar puncture—clear cerebrospinal fluid under increased pressure (13 mm.); Wassermann test negative, and cell count was 6 cells per c.mm. X-ray (Doctor G. W. Welton)—"bony defect of irregular size and apparently of one inch in diameter in the lower posterior area of left parietal bone at its junction with the left occipital bone; no linear fracture observed."

Treatment.—The presence of the definite signs of an increased intracranial pressure persisting over this period of 8 months since the injury would indicate that a chronic cerebral edema is present and the treatment should be directed toward a lessening of this increased intracranial pressure in the hope that the condition of the patient can be improved—particularly the headache, the signs of cortical irritation, and thus the great danger of convulsive seizures be lessened; naturally, it is very doubtful if the vision can be improved—the lesion being a central cortical one of the left occipital lobe and the median portion of the right occipital lobe due to the penetration of the iron bolt through these cerebral tissues and their optic radiations. The patient, therefore, was advised a non-proteid and non-stimulative diet (no meat, meat-soup, tea, coffee or alcohol), an inactive life with little excitement and worry, daily catharsis and general hygienic measures for a period of 3 months in the hope that the increased intracranial pressure would then be lowered. Patient was discharged with these instructions on the eighth day after admission.

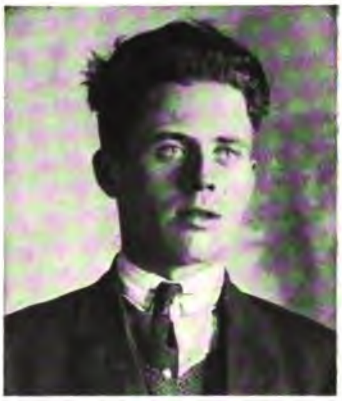

FIG. 128.—The normal physical appearance of this patient contrasts strikingly with his most serious cerebral injury of the left occipital lobe and its characteristic visual impairment of hemianopsia.

Last Examination (September 14, 1919—12 months after last examination and 21 months after injury).—Patient says, "I am possibly less restless but I am still having trouble"; headaches persist, associated with dizzy spells and an occasional loss of consciousness of momentary duration; no convulsive seizures, however. No improvement of vision. Site of former operation still tense with slight pulsation palpable. The physical and neurological findings are practically the same as at the former examination one year ago, while the pressure of the cerebrospinal fluid at lumbar puncture remains at 13 mm. as registered by the spinal mercurial manometer.

Treatment.—In order to lessen this increased intracranial pressure and thus not only improve the patient's general condition but to decrease the danger of convulsive seizures, a left subtemporal decompression was advised—even at this late date. Patient, however, refused his consent for the operation at this time and wants "to think it over"; the expectant palliative treatment, as outlined above, was therefore continued.

Remarks.—It is unfortunate that a left subtemporal decompression and drainage was not performed at the time of the removal of the depressed area of bone; there must have been a high intracranial pressure at that time due to the intradural hemorrhage and to the cerebral edema resulting from the cerebral trauma, and it is impossible to relieve high intradural pressure satisfactorily in these patients through a small dural opening at the site of the depressed area of bone; not only is it distinctly dangerous to attempt to do so for fear of operative damage to the underlying cerebral tissues and thus producing a definite cerebral impairment clinically, but it is impossible through the small dural defect to secure adequate decompression and drainage—and it is impossible if the dura is not widely opened. All of these patients having depressed fractures of the vault and associated with marked signs of increased intracranial pressure should have an ipsolateral subtemporal decompression first, and then the local depressed area of bone can be elevated, removed, and the other necessary operative procedures be accomplished safely with little or no danger to the underlying cerebral cortex.

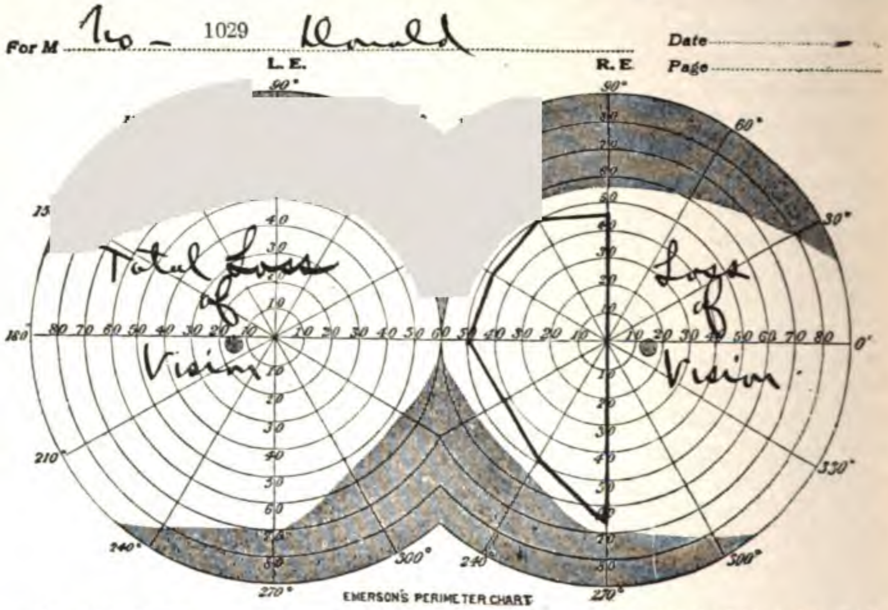

FIG. 129.—Complete loss of vision of the left eye and of the temporal half of the field of vision of the right eye following a depressed fracture and penetrating injury in the left parieto-occipital area, indicating a lesion of the left occipital lobe or its optic radiations posterior to the optic chiasm to cause a right homonymous hemianopsia and a lesion of the mesial surface of the right occipital lobe or of its particular optic radiations to cause a unilateral loss of the nasal half of the field of vision of the left eye. The consensual pupillary light reflex was normal in each eye. It is possible, however, that in this patient there was, in addition to the injury of the left occipital lobe producing the right homonymous hemianopsia, a direct injury to the left optic nerve which would account for the total loss of vision of the left eye (a primary optic atrophy); the normal consensual pupillary light reflex would then be puzzling, although it would be possible for it to be present.

The complete loss of vision of the left eye and of the temporal [redacted] the field of vision of the right eye, particularly in the absence of a p or secondary optic atrophy and in the presence of the normal pu reflex of both eyes, would indicate that the visual lesion here is a cortical one of the entire left occipital lobe and of the median portio right occipital lobe—that is, the iron bolt had penetrated through occipital lobe, the falx cerebri and the median portion of the right lobe. The existence of the normal pupillary reflex, and especially the consensual light reaction, indicates that the visual lesion must be posterior to the optic chiasm and thus, in this type of cranial injury, the lesion is also posterior to the corpora quadrigemina and therefore involving the cortex of the occipital lobe as described above and their subcortical optic radiations. The necessity for the subtemporal decompression and drainage immediately following the injury need not be emphasized when we consider the amount of hemorrhage and cerebral edema which must have been associated with such a severe cerebral trauma; the treatment should be similar to gunshot injuries of the brain when the dura has been penetrated and a direct local cerebral damage has resulted. The history that this patient could see out of the left eye for several hours following the injury is indicative of an increasing cortical hemorrhage and cerebral edema.

It will be very interesting to follow this patient over a period of months and even years in order to ascertain the ultimate result—with or without operation. The great risk naturally is the onset of convulsive seizures, particularly in the presence of a definite cortical and cerebral lesion associated with an increased intracranial pressure; these two factors are the chief causes of traumatic epilepsy and their influence should be lessened as much as possible—the damaged cerebral cortex cannot be regenerated, but the increased intracranial pressure can be lessened so that the cerebral cortex as a whole will be less irritable and in a more resistant condition and thus the onset of major convulsive seizures will be postponed and even entirely avoided; the spells of momentary loss of consciousness (*petit mal* attacks) can also be lessened so that they may not recur. The fact that the expectant palliative treatment has failed to decrease the increased intracranial pressure entirely and down to its normal level—this fact emphasizes the advisability of a mechanical relief of this increased intracranial pressure by means of a left subtemporal decompression.

B. Chronic Brain Injuries Associated with a Fracture of the Base of the Skull

It has been repeatedly emphasized in this book that it is of comparatively little or no importance whether the vault or the base of the skull is fractured or not at the time of the acute cranial injury, and this same opinion is true regarding the presence or not of a fracture of the base in chronic brain injuries. It is of interest in these patients for the physician to realize that the original trauma was of sufficient force to produce a fracture of the base of the skull, but this positive knowledge does not in any degree influence the treatment or the prognosis of conditions of chronic brain injuries.

Of the greatest importance in these patients, however, is the presence or not of an increased intracranial pressure; if present, then its lowering by means of the cranial operation of subtemporal decompression, which also permits drainage of the usual condition in these patients of a "wet," edematous condition of the brain—a true chronic cerebral edema, and thus a permanent improvement is possible; if there is no increase of the intracranial pressure, then there is little, if anything, that can be offered to improve the condition of these patients—the cerebral damage having already occurred, whether due to cortical lacerations, severe contusions, etc. Many of the post-traumatic conditions of these patients could have been prevented—if not wholly, then partially—by the proper treatment within a short time after the acute cranial injury, which is the ideal time to obtain the best results possible; at the late date of months or years after the injury, the greatest possible benefit may be merely a slight improvement.

B. *Old brain injuries associated with fractures of the base of the skull. No operation. Symptoms and signs persisting. Operation advisable.*

Case 101.—Old severe brain injury associated with a fracture of the base of the skull and with signs of an increased intracranial pressure; partial secondary optic atrophy. No operation. Symptoms and signs persisting. Operation advisable.

No. 150.—Andrew. Thirty-seven years. White. Married. Carpenter. Scotland.

Admitted March 28, 1914—6 months after injury. Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Discharged April 10, 1914—12 days after admission. Operation refused.

Family history negative.

Personal History.—Always well and strong. On September 30, 1913 (6 months ago), scaffold upon which this patient was working collapsed and the patient fell a distance of 45 feet, striking upon the back of his head; immediate loss of consciousness; profuse bleeding and discharge of "watery" fluid from the left ear; remained in a hospital for a period of one month. Since then, he has had continuous headaches, dizziness upon stooping, impairment of vision particularly of left eye, increasing irritability and faulty memory for recent events; during the past month, the severity of the headaches has increased so that the patient comes to the hospital for treatment.

Examination (6 months after injury).—Temperature, 99.8°; pulse, 74; respiration, 18; blood-pressure, 130. Rather poorly nourished; anxious facies. Severe frontal and bitemporal headaches; restless and very irritable. Cannot remember correctly anything that happened during the 2 years before the accident and also since the injury. Marked tremor of both hands. No external signs of cranial injury. No paralysis of extremities or sensory impairment of any kind. Speech fair; no definite slurring; no aphasia or paraphasia. Hearing—left ear definitely impaired; Weber's test—sound referred always to left ear; Rinne's test—air conduction greater than bone conduction in both ears. Pupils—left larger than right and reacts to light sluggishly. Reflexes—patellar very much exaggerated, there being both an exhaustible patellar and ankle clonus; no true Babinski but no plantar flexion on right foot; abdominal reflexes—right more active than left. Fundi—retinal veins dilated; definite secondary optic atrophy—more marked in left eye where there persists a large amount of pigment over the macula lutea (the result of a former hemorrhage of the central retinal artery). Visual fields—left very much contracted but concentrically. Visual acuity—right 9/10; left 1/100 (being almost completely blind in this eye). Lumbar puncture—clear cerebrospinal fluid under increased intracranial pressure (approximately 13 mm.); Wassermann test negative and cell count was 6 cells per c.mm. X-ray (Doctor A. J. Quimby)—"small linear fracture extending vertically downward through the left squamous bone toward the left mastoid area; no other abnormalities seen.''

Treatment.—The fact that the headaches were increasing in severity and the presence of an increased intracranial pressure, in spite of excellent medical treatment, would tend to make the operation of subtemporal decompression advisable; if after a period of 3 months, however, of most careful medical treatment directed toward the lowering of this increased intracranial pressure and if it should then fail, it would be advisable to perform a subtemporal decompression and thereby not only lower mechanically the increased intracranial pressure and cause a cessation of the headache, but it would lessen the cortical irritability and thus the danger of convulsions would be avoided. Naturally, if an operation must be performed at this late date, it would have been much better surgical judgment to have operated immediately after the injury—as soon as the symptoms and signs of shock had disappeared and after the signs of an increased intracranial pressure had appeared.

At the end of a three months' medical treatment consisting chiefly of general hygiene, very light diet—no meat, meat-soup, tea, coffee or alcohol in any form, and only small portions of fish, eggs, vegetables, milk, water (so that the patient was practically always hungry), daily catharsis and warm baths—in spite of this treatment the condition of the patient was practically the same as at the preceding examination; a left subtemporal decompression was therefore advised but the patient refused his consent.

Examination (June 20, 1916—33 months after injury).—Patient still complains of headache but not of such severity as during the year following the injury; very irritable, however, so that his wife left him 8 months ago. Memory has improved somewhat, but ''it hurts to think hard.'' Physical examination is practically the same as at the last examination except for the presence of a definite right Babinski and a lessening of the visual acuity of the right eye which is now only 6/10, whereas before it was 9/10. Patient is unable to work so vigorously as formerly and seems to have lost all ambition to do so. The danger of an operation, however, is far greater, he thinks, than his impairments.

Last Report (July 12, 1918—62 months after injury).—Sister writes that ''Andrew has never been himself since the accident; he no longer complains as before but remains by himself; seems stupid.''

Remarks.—An operation performed early would, I believe, have prevented the gradual and continuous mental and emotional deterioration of this patient; it is very doubtful whether the vision of the left eye could have been benefited by an operation, as its impairment was primarily due, apparently, to a hemorrhage of the retinal artery in the left optic nerve itself; the visual impairment was also due to the increased intracranial pressure by its causing a mild secondary optic atrophy and would have been the same as in the right eye, which even at the last examination was only impaired to the extent of 6/10. The left middle ear impairment was only sufficient to lessen its hearing comparatively, in that the air conduction was still greater than the bone conduction but only to a less extent than in the right ear.

If permission for the operation could have been obtained, it would have been very interesting to have observed this patient carefully and thus note any marked consequent change and improvement, so that future patients of the same character could be competently advised as to their treatment.

Case 102.—Old severe brain injury associated with a fracture of the base of the skull and signs of an increased intracranial pressure; convulsive seizures; no operation. Symptoms and signs persisting. Operation refused.

No. 111.—Otto. Twenty-nine years. White. Single. Clerk. Germany.

Admitted March 1, 1915—4 years after injury. Polyclinic Hospital. Referred by Doctor E. W. Lee.

Discharged March 11, 1915—10 days after admission. Operation refused.

Family history negative.

Personal History.—Always well and strong. Four years ago, patient fell down the cellar stairs, landing upon his head; immediate loss of consciousness; treated at home expectantly. Profuse hemorrhage from both ears, mixed with a "watery fluid," ceased after 3 days; on the sixth day after injury, the patient had a severe general convulsion lasting 8 minutes but no localizing signs were observed; he remained in bed for 2 weeks and was able to work 3 weeks after injury. Since then, however, patient has had severe headaches and a general convulsion has occurred once a month until one year ago, when the convulsions ceased but the headaches continued as before. The treatment consisted of the usual medical and general hygienic measures. Two days ago, another general convulsion occurred, to be followed by a similar attack yesterday; the patient walked into the hospital to obtain relief of headaches and to ascertain if anything could be done for the convulsive seizures.

Examination upon admission (4 years after injury).—Temperature, 98.6°; pulse, 76; respiration, 20; blood-pressure, 128. Patient apparently normal except for the complaint of continuous headaches and the history of convulsive seizures; tongue shows several old lacerations due to its being bitten during the "spells." No external signs of cranial injury. No motor or sensory impairments. Hearing negative; otoscopic examination negative. Pupils equal and react normally. Reflexes—patellar very much exaggerated but equal; double exhaustible ankle clonus and double suggestive Babinski; abdominal reflexes depressed. Fundi—retinal veins enlarged; general congestion and redness of both retinæ; nasal halves of both optic disks definitely obscured by edema, whereas temporal halves were fairly clear and distinct. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 14 mm.); Wassermann test negative and cell count was 9 cells per c.mm. X-ray—"negative for fracture."

Treatment.—Owing to the signs of increased intracranial pressure as shown by the ophthalmoscopic examination and the measurement of the cerebrospinal fluid at lumbar puncture, it was thought advisable to p
patient upon strict hygienic treatment with the vigorous use o
bromides in the hope that the convulsive seizures could be prevent
period of at least 3 months, and then if the signs of an increased
were still present, it could be fairly definitely stated that the
intracranial pressure was due to a chronic cerebral edema f
former brain injuries (whether associated with intracranial

not), and that it was not due to the "wet," edematous condition of the brain following the frequent convulsions; that is, the increased intracranial pressure was a factor in causing the convulsions, and not the convulsions being the cause of a "wet," edematous brain and therefore the cause of the increased intracranial pressure—that is, the increased intracranial pressure was primary and the convulsions were secondary rather than the convulsions being primary and the increased intracranial pressure being secondary as the result of the convulsions.

At the end of this medical treatment for three months, however, during which period no convulsions had occurred, there were still present the signs of an increased intracranial pressure as revealed in the fundi and in the measurement of the pressure of the cerebrospinal fluid at lumbar puncture, and thus indicating that the increased intracranial pressure was not the result of the convulsions causing the cerebral edema but that the cerebral edema was a factor in producing the convulsions, which during this period of 3 months had been controlled by the liberal use of triple bromides. It was, therefore, considered advisable to advocate a subtemporal decompression as the mechanical means of lessening this increased intracranial pressure and thereby lower the cortical irritability, so that the convulsions would be less liable to occur—naturally this "operation" only being advised when the medical treatment had failed to lessen this increased intracranial pressure. The patient, however, considered the risk of the operation too great and refused; he was, therefore, put upon a strict medical treatment in the hope that the condition could be benefited.

Examination (April 18, 1917—73 months after injury).—By the vigorous use of triple bromides and luminal, patient has been able to control the convulsions so that they occur only once every 2 or 3 months; he has, however, noticeably deteriorated both mentally and emotionally, and although he is able to work, yet (as his brother states) his employers merely "tolerate" him in the office and he is given no responsibility. The physical examination is practically the same as at the examination 25 months ago—the signs of an increased intracranial pressure being still present.

Treatment.—After this long period of convulsive seizures it is exceedingly doubtful whether any treatment—operative or otherwise—could result in a "cure" of this patient; that is, the irritability of the cortex had probably reached such a degree from the prolonged increase of the intracranial pressure and the other irritating factors as a result of so many preceding convulsions, that the formation of the so-called epileptic "habit" is permanently established. Relatives desired the operation now but the patient obstinately refused "to have my head cut."

Last Report (October 2, 1918—91 months after injury).—In a letter, brother states that the patient is in practically the same condition as at the last examination; works, however, now only 2 days a week and seems to be losing confidence in himself; no longer complains of headache; only leaves the house when it is absolutely necessary.

Remarks.—This gradual mental and emotional deterioration, which practically always follows convulsive seizures persisting over a long period of time and particularly when associated with an increased intracranial pressure, is most pitiful to observe; the risk of a simple decompression operation is so slight compared with the almost certain eventual mental and emotional deterioration that the operation is, in fact, no risk at all. Naturally, the longer the convulsive seizures have persisted in the presence of an increased intracranial pressure, and the more frequent and more severe the convulsions have become, the less hopeful is the prognosis following any treatment—operative or otherwise—and for this very reason an increased intracranial pressure complicated by convulsive seizures should be relieved as early as possible in order to facilitate a permanent recovery, if it can be obtained.

It would seem that this patient will follow the path of other patients similarly affected, in that the mental and emotional deterioration will progress gradually—a little more each year, until the patient leads merely a vegetative existence so far as his usefulness to the community is concerned, and then, as the convulsions become more frequent, he thus becomes a greater and greater burden to his friends and relatives, and is finally committed to an asylum where he dies eventually in the condition of status epilepticus or from intercurrent disease. Surely an early attempt to lessen the increased intracranial pressure—if medical treatment has failed—and thus give the patient a chance of recovery, would clearly be indicated, and even if in many of the older cases the ultimate end-result is the same and the condition would seem to have been merely delayed, yet in those patients treated early who do make an excellent recovery, they are thus spared this later institutional life and the effort is justified.

C. CHRONIC BRAIN INJURIES ASSOCIATED WITH A DEPRESSED FRACTURE OF THE VAULT WITH PERSISTING SYMPTOMS AND SIGNS; MINOR AND MAJOR EPILEPSY. OPERATION.

In the treatment of these selected patients having old depressed fractures of the skull and with symptoms and signs persisting, the presence or not of an increased intracranial pressure is of the greatest importance. If there is no marked increase of the intracranial pressure present, then a simple elevation or removal of the depressed area of bone may be, and frequently is, sufficient to secure a good result; if, however, there is present a definite increase of the intracranial pressure, then in selected patients a simple subtemporal decompression will suffice and the local depressed area of bone need not be removed, as it may not in itself be a sufficient cause of the cortical irritation, and especially, if it is adjacent to the longitudinal or occipital sinuses, its safe removal would be a difficult one technically; in other patients in the presence of a high intracranial pressure, it is essential in order to obtain an improvement at the least risk, to perform a subtemporal decompression first and then to elevate or remove the depressed area of bone. In old doubtful cases, and especially of this character, it is better surgical judgment to perform a subtemporal decompression first, and if a definite improvement does not result within a period of weeks, or, at
months, then to elevate or remove the depressed area of bone which
the local cortical irritant.

C. *Old brain injuries associated with depressed fractures of t* *no operation; symptoms and signs persisting; minor and major epilepsy. Operation. Excellent recovery.*

a. Removal of depressed area of vault alone—only mild signs of an increased intracranial pressure being present.

CASE 103.—Old brain injury associated with a depressed fracture of the vault of the skull; symptoms and signs persisting. Removal of depressed bone. Excellent recovery.

No. 110.—Charles. Thirty-eight years. White. Married. Steam-fitter. United States.

Admitted March 31, 1914—7 months after injury. Polyclinic Hospital. Referred by Doctor F. N. Noble.

Operation April 3, 1914. Removal of depressed area of bone.

Discharged April 15, 1914—12 days after operation.

Family history negative.

Personal History.—Always well and strong. Seven months ago while at work, patient was struck upon the head by a fire-brick, falling a distance of 8 stories; loss of consciousness for several moments; no bleeding from nose, mouth or ears; lacerated wound over the upper left occipital area. Patient was able to walk to a hospital where he remained 4 days and was then discharged with a sterile bandage over the laceration of the scalp; he, however, was obliged to return to the hospital the same day on account of nausea and vomiting. He was again discharged at the end of 7 days, but since that time he has had continuous frontal headaches, and complains of being in a sort of "cloud" at times—dazed and drowsy; no convulsions; hearing of left ear is more impaired since the injury. Unable to work.

Examination upon admission (7 months after injury).—Temperature, 99°; pulse 78; respiration, 18; blood-pressure, 144. Well-developed and nourished. Over the left occipital area was a small sinus discharging a thin purulent material; careful probing revealed a depressed fracture of the underlying vault. No motor or sensory impairment. Pupils equal and react normally. Reflexes—patellar exaggerated, right more than left; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi—retinal veins dilated; nasal margins of both optic disks blurred; physiological cups of both optic disks shallow from the new tissue formation. Lumbar puncture —clear cerebrospinal fluid under slightly increased pressure (approximately 11 mm.); Wassermann test negative and cell count was 7 cells per c.mm. X-ray (Doctor A. J. Quimby)—"depressed fracture of left occipital bone—diameter about 2 cm."

Treatment.—As the intracranial pressure was not markedly increased, merely a removal of the depressed area of bone was advised, both to stop the infective process and to prevent not only an extensive osteomyelitis but also a meningitis from resulting, and to lessen the danger of intradural complications, particularly convulsive seizures.

Operation (7 months after injury).—Removal of depressed area of vault: an S-shaped incision through site of old laceration of the scalp and the discharging sinus over the left occipital bone; upon retracting the scalp, pieces of hair and dirt were found buried deep in the depression and undoubtedly the cause of the suppuration. A comminuted depressed area of underlying bone, the size of a silver quarter, was exposed and the depressed fragments removed by enlarging the bony opening with rongeurs to a size of a silver half-dollar; all necrosed bone was removed. Much granulation tissue upon the underlying dura (possibly due to a former extradural hemorrhage and infection); dura itself not tense and had not been torn, so that naturally it was not opened, both from the great danger of extending the infective process and on account of the absence of intradural pressure. Usual closure with 3 drains of rubber tissue inserted. Duration, 25 minutes.

Post-operative Notes.—Uneventful operative recovery; no signs of meningeal irritation occurred.

Examination at discharge (12 days after operation).—Temperature, 98.6°; pulse, 80; respiration, 22; blood-pressure, 142. No complaints except for slight soreness at the site of operation; no headache. Wound has healed *per primam.* Reflexes—patellar very active, right still greater than left; otherwise negative. Fundi—retinal veins rather full but less so than before operation; blurring of nasal margins of both optic disks persists.

Examination (June 23, 1914—3 months after operation and 10 months after injury).—No complaints; "as well as ever." Works daily. Operative wound has healed perfectly and slight pulsation palpable. Reflexes—less active than before and practically equal. Fundi—retinal veins enlarged but no definite blurring of nasal margins of either optic disk.

Examination (December 10, 1916—40 months after injury).—No complaints; at work daily. Reflexes active but otherwise negative. Fundi negative. Operative wound is being filled in with new bone formation.

Last Report (October 10, 1918—62 months after injury).—Letter from patient states that he is "as well as ever"; no headache, works daily.

Remarks.—It is in depressed fractures of the vault that röntgenograms are most important in the treatment of cranial injuries; even though careful palpation and, if there is an overlying laceration of the scalp, then careful probing, do not reveal an underlying fracture and, more important, a depressed fracture, it is then that an X-ray picture taken at the appropriate angle is of such valuable aid in the treatment of cranial injuries; for if a depressed fracture of the vault is present, then an early elevation and usually the removal of the depressed area of bone is necessary to lessen the danger of future complications—particularly an irritable cerebral cortex and the resulting convulsive seizures.

In this patient, an early local operation to remove the depressed area of bone would very probably have prevented the infective process and thus the patient would have been spared the serious complication of a resulting osteomyelitis and even a meningitis itself. If the dura had been torn at the time of the injury, it is difficult to conceive how this patient could have escaped the serious complication of a meningitis, and therefore an operative procedure is almost obligatory when there is a depressed fr of the vault in the presence of an overlying laceration of the scalp absence of marked signs of intracranial pressure obviated the n of performing a subtemporal decompression first, although in doubt it is always the safer procedure.

CASE 104.—Old brain injury associated with a depressed frac vault and with mild signs of an increased intracranial pressure; symptoms and signs persisting. Removal of bony depression. Excellent recovery.

No. 190.—Tom. Twenty-there years. White. Mechanic. U. S.

Admitted December 13, 1914—8 months after injury. Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Operation December 15, 1914. Removal of depressed area of vault.

Discharged December 24, 1914—9 days after operation.

Family history negative.

Personal History.—Always well and strong. While riding upon a wagon, patient was jolted off and fell headlong into a stone curbing; immediate loss of consciousness; no bleeding from nose, mouth or ears; taken to the Polyclinic Hospital, where he remained until May 14 (16 days after injury); patient refused at that time the operation to elevate or remove the depressed area of the left frontal bone, so that only the laceration of the scalp over the left frontal area was sutured loosely after being thoroughly cleansed. Upon his discharge from the hospital, patient had almost daily headaches; no spells of dizziness or convulsions. Headaches have continued during the past 8 months associated with mild signs of increased intracranial pressure; patient is not so alert mentally as formerly; a definite irritableness has also appeared during the past two months.

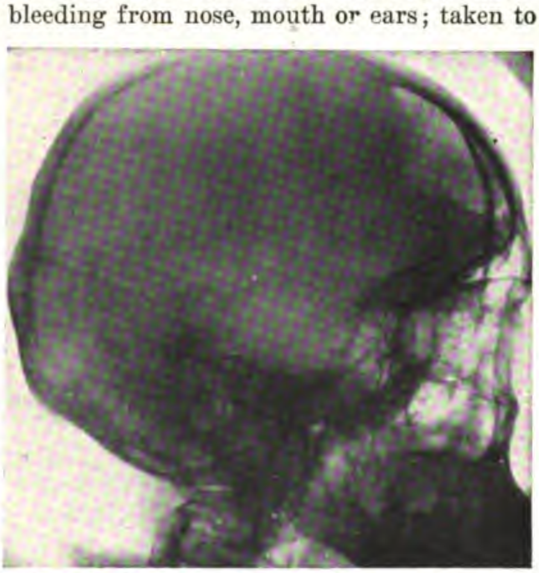

FIG. 130.—Huge bony defect of the left frontal bone, due to the removal of an extensive depressed fracture, producing continuous headache associated with mental retardation and emotional instability. Excellent recovery.

Examination upon admission (8 months after injury).—Perfectly healed scar over left frontal region; palpation reveals a definite depression of the underlying bone. No motor or sensory impairments. Pupils equal and react normally. Reflexes negative. Fundi—retinal veins rather full; nasal margins of both optic disks and nasal halves of left optic disk distinctly blurred by edema. Lumbar puncture—clear cerebrospinal fluid under slightly increased pressure (approximately 11 mm.); Wassermann test negative and cell count was 4 per c.mm. X-ray (Doctor A. J. Quimby)—"oval depression, 2 inches in diameter, of left frontal bone; no radiating fractures."

Treatment.—A local removal, or if possible, an elevation of the depressed area of bone advisable to prevent future complications, particularly the great danger of convulsive seizures later in life, and also to lower possibly the very mild increased intracranial pressure; if the increased intracranial pressure were higher, then a subtemporal decompression would be advisable first, to be followed by a removal of the depressed area of bone. The parents were finally convinced of the necessity and really the safety of this operative procedure.

Operation (8 months after injury).—Removal of depressed area of bone: curvilinear incision of 2½ inches made over the left frontal region extending to the median line; upon retracting the scalp, the underlying depression was ascertained and a small trephine opening made at the outer portion of the depressed area; an attempt to elevate the depressed bone was not successful, so that the bone itself was entirely rongeured away even beyond the longitudinal sinus in the midline over which it extended; no complications. Underlying dura now became convex; it was not markedly tense and not torn and naturally no opening was made. Usual closure with 2 drains of rubber tissue inserted. Duration, 30 minutes.

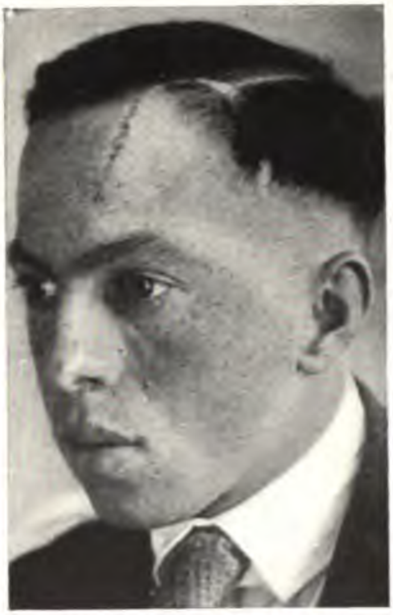
FIG. 131.—Sixteen months after the removal of a large depressed area of the left frontal bone, causing definite symptoms and signs. Excellent recovery with no complaints.

Post-operative Notes.—Uneventful operative recovery, so that patient was discharged 9 days after operation; incision healed *per primam.* X-ray picture discloses the bony defect of the left frontal area (Fig. 130).

Examination (April 10, 1916—16 months after operation).—No complaints; works daily; stands well in his class in evening school. Reflexes negative. Fundi negative. Operative area slightly depressed and pulsates normally (Fig. 131).

Last examination (November 6, 1918—55 months after injury and 47 months after operation).—No complaints; works daily and is considered a competent mechanic. Operative area slightly depressed and the edges are being filled in with new bone formation; only slight pulsation palpable. Reflexes negative. Fundi negative.

Remarks.—It is possible that this patient, even if no operation had been performed, might never have experienced any ill-effects from this bony depression other than possibly slight headaches; but when we consider the frequency of convulsive seizures occurring in adults long after the cranial injury producing the bony depression in youth, and then the doubtful prognosis in these patients, even with an operation, there is no question as to the advisability of elevating and removing all depressed fractures of the vault for fear of future complications; once the convulsions do occur, then the patient can never be assured that they will cease upon the removal of the irritative cause. It is, therefore, commonly acknowledged that depressed fractures of the vault should be elevated or removed; the dang of the operation itself is slight and as the dura need not be opened in the vast majority of these patients, the operation can scarcely be considered a major operation—in fact, it is not.

It is rarely possible to elevate the depressed area of bone, unless in children and shortly after the injury; in adults and in children after a period of several weeks, the depressed bone becomes so firmly "fixed" and ankylosed in its depressed position, that to elevate it forcibly would in most patients be a dangerous procedure and far more difficult than to remove it; the resulting deformity is usually slight, being in most patients within the hair-line, and even if it does cause a noticeable depression, yet that phase of the condition cannot be considered an important factor; the use of silver or bone plates subcutaneously is to be most strongly condemned—merely the insertion of possible complications.

b. *Decompression alone—there being present definite signs of an increased intracranial pressure.*

CASE 105.—Old severe brain injury associated with a depressed fracture of the skull and with definite signs of an increased intracranial pressure; convulsive seizures and hemiplegia. Right subtemporal decompression. Improvement.

No. 090.—Margaret. Twenty-two years. White. Single. Maid. U. S.

Admitted December 1, 1913—6 years after injury. Polyclinic Hospital. Referred by Doctor L. B. Rogers.

Operation December 2, 1913. Right subtemporal decompression.

Discharged December 10, 1913—8 days after operation.

Family history negative.

Personal History.—Always well and strong. Six years ago while walking along the pavement, patient was struck over the left parietal area of the head by a stone-tile weighing 25 pounds and falling from a height of 4 stories; immediate loss of consciousness; taken to Bellevue Hospital. Forty-eight hours later, when patient became conscious, it was ascertained that the right side of body was paralyzed, associated with an incomplete motor aphasia; laceration of the scalp over the left parietal area was merely sutured. Two months after the injury, the depressed area of left parietal bone was elevated and within ten days there was a marked improvement of the right hemiplegia and speech impairment. Three years after injury (3 years ago), patient began having Jacksonian convulsions limited to the right arm, right leg and also slightly to the right side of face, but no loss of consciousness occurred; these convulsive seizures occurred once each night and at the end of 3 months, patient was again operated upon in Bellevue Hospital and the former depressed area of the left parietal bone was removed; dura, however, was not opened; after this operation, the speech improved still more but the paralysis of the right side of body remained practically the same with no improvement of the convulsive seizures. During the past 2 years, she has complained more and more of continuous headaches, almost daily convulsions beginning in the right arm and then becoming general with loss of consciousness and of sphincteric control, a definite impairment of vision and increasing drowsiness so that the patient is no longer able to work.

Examination upon admission (6 years after injury).—Temperature, 98.6°; pulse, 84; respiration, 20; blood-pressure, 134. Well-developed and nourished. Mental retardation and unstable emotionally. Over left parietal area was an irregular bony defect not larger possibly than a silver dollar; no pulsation palpable. Right spastic paralysis; right heel does not touch the ground; no sensory impairment. Hearing negative. Mentality definitely clouded and the patient is emotionally dulled. Pupils equal and react normally. Reflexes—patellar very much exaggerated, right much more active than left; right ankle clonus and right Babinski; abdominal reflexes depressed. Fundi—retinal veins dilated; both optic disks rather whitish with irregular and shallow physiological cups; nasal margins blurred; both retinæ suffused and congested throughout, having a reddish "pepper-pot" appearance. Lumbar puncture—clear cerebrospinal fluid under a definite increase of pressure (approximately 16 mm.); Wassermann test negative and cell count was 5 cells per c.mm. X-ray (Doctor A. J. Quimby)—"irregular bony defect of almost 2 inches in size over upper left parietal area; new bone formation at periphery" (Fig. 132). Definite speech impairment easily elicited by the usual speech tests—not a true motor aphasia but a paraphasia of mild degree.

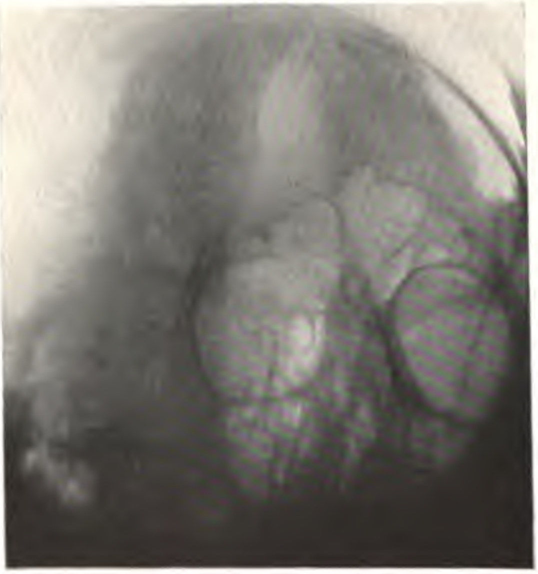
FIG. 132.—Irregular bony defect (but dura unopened) of large diameter over the left parietal area, in a patient having a right hemiplegia with Jacksonian convulsive seizures. A marked improvement followed a subtemporal decompression.

Treatment.—From the fact that there were still present definite signs of an increased intracranial pressure and thus a definite organic cause for the headache and the mental and emotional impairments, it was considered advisable to perform a subtemporal decompression to lessen this increased intracranial pressure, and thereby lower the cortical irritability so that the convulsive seizures might be decreased in number and severity; on account of the definite increase of the intracranial pressure, it was thought better surgical judgment to perform a right subtemporal decompression rather than a left subtemporal de pression for fear of damaging the motor speech area of the left cere cortex (patient being right-handed); as we know, however, that the of this resulting impairment would have been practically nil, so that it is realized that a left subtemporal decompression could and should been performed upon this patient, as it would have lessened equa the general intracranial pressure and also decreased the pressure directly over the affected area of the left cerebral cortex much more efficiently.

Operation (6 years after injury).—Right subtemporal decompression: usual vertical incision, bone removed and no complications. Dura very tense and upon incising it, clear cerebrospinal fluid spurted a distance of ½ inch—revealing a very "wet," edematous cortex under pressure; upon enlarging the dural opening, much cerebrospinal fluid escaped and thus the intradural pressure was quickly lessened and a rupture of the cerebral cortex was avoided; at the end of the operation, the brain receded and pulsated normally. Dura was very vascular with many newly-formed vessels throughout so that it oozed freely. Arachnoid was very cystic with much induration about the vessels in the sulci. Usual closure with 2 drains of rubber tissue inserted. Duration, 50 minutes.

Post-operative Notes.—Uneventful operative recovery; incision healed perfectly and patient was discharged 8 days after operation—no convulsions having occurred.

Examination (June 4, 1914—6 months after operation).—No convulsions have occurred; occasional headache, however, and "heaviness" in the head upon stooping; patient says she can see better—"no longer a cloud present." Decompression area bulges slightly beyond flush of scalp; normal pulsation visible. Spasticity of right arm and leg less marked than before operation; right ankle clonus and Babinski persist. Fundi—the signs of secondary optic atrophy are present as before operation, but the blurring along the nasal margins and the dilatation of the retinal vessels have disappeared.

Examination (October 20, 1916—33 months after operation).—Patient had her first convulsion 10 months after the operation; since then, she has had one convulsion about every 3 months. Patient no longer complains of headaches, however, and is able to work; impairment of speech has lessened and, with the exception of the convulsions, patient has improved in every way. Decompression area flush with the surrounding scalp and pulsates normally.

Last Report (November 2, 1918—59 months after operation).—Sister writes that patient has a general convulsion every 2 to 3 months, but that "they no longer upset her because she can get right up and work afterward." Paralysis of right arm and leg better than before the operation; speech also definitely improved; only occasional headaches.

Remarks.—The fact that the convulsive seizures have returned and even though they are less frequent and possibly less severe than before the operation, yet the fact that they have returned in spite of the lessened cortical irritability merely means, I fear, that the end-result of frequent convulsions and eventually epileptic dementia have only been delayed; this was to have been expected when we consider that this patient had had convulsions following a depressed fracture of the skull for a period of 3 years before the decompression operation, and that they had been so frequent as to occur daily; the presence also of a right hemiplegia indicating a definite damage to the underlying left hemisphere would tend to confirm the presence of a definite organic damage to the left cerebral cortex itself and therefore there was present in this patient a local source of cortical irritation, so that even after the increased intracranial pressure had been relieved, there would still be a definite cause for convulsive seizures to occur. It would have been better surgical judgment to have performed a left subtemporal decompression and exploration first, and if the local cortical irritation could have been removed, to have done so, and thus this patient would have been given a still greater chance of ultimate recovery. Even if the condition of this patient was to be benefited only for a period of several years, yet the operation was justified both from the patient's standpoint, the relatives' and the community's—even in the economic factor alone.

CASE 106.—Old severe brain injury associated with a depressed fracture of the vault and with signs of an increased intracranial pressure; mild left hemiplegia with later convulsive seizures. Right subtemporal decompression. Improvement.

No. 482.—Lizzie. Twenty-two years. White. Single. Operator. U. S. Admitted January 2, 1916—17 years after injury. Polyclinic Hospital. Referred by Doctor W. B. Pritchard.

Operation January 10, 1916. Right subtemporal decompression.

Discharged January 21, 1916—11 days after operation.

Family history negative.

Personal History.—When patient was 5 years of age (17 years ago), she fell from a second-story window, striking her head upon the ground; immediate loss of consciousness and paralysis of entire left side of body; she remained in a hospital for 6 weeks and at discharge the left arm and left leg were still definitely weak. Gradual improvement occurred, however, and the patient was considered a normal child except for a slight weakness and awkwardness of the left arm and left leg. Five years ago, a dull aching pain began in both the left arm and left leg, and one month later, frontal and occipital headaches occurred almost daily and have continued with increasing severity and frequency up to the present time, so that she has been unable to work during the past year; during the past 12 months she has had 7 general convulsive seizures with loss of consciousness.

Examination upon admission (17 years after injury).—Temperature, 98.6°; pulse, 80; respiration, 22; blood-pressure, 128. Well-developed and nourished. Over the right parietal area is a slight irregular bony depression extending downward below the attachment of the right temporal muscle to the parietal crest. Slight weakness of both the left arm and left leg, as demonstrated by the hand-grip and by testing the strength of each leg; left leg quickly becomes tired after walking several blocks or after standing a few minutes. No sensory impairment. Hearing negative. Mentality—definite retardation and emotionally unstable—very irritable and "loses her temper" upon the slightest provocation. Pupils equal and react norma
Reflexes: patellar—left greater than right; exhaustible left ankle clo
and absence of left plantar reflex with a tendency to a left Babins
abdominal reflexes—left difficult to elicit. Fundi—retinal veins slig
enlarged; lower nasal quadrants of both optic disks indistinct and
nasal margins slightly blurred and irregular from new tissue format
right physiological cup shallow. Lumbar puncture—clear cerebrosp

fluid under increased pressure (20 mm.); Wassermann test negative and cell count was 5 cells per c.mm. X-ray (Doctor W. H. Stewart)—"definite periostitis over right parietal bone, which was slightly depressed for an area of almost 3 inches in diameter, extending down to the right squamous bone."

Treatment.—The definite signs of an increased intracranial pressure as revealed both by the ophthalmoscope and by the lumbar puncture, the presence of a distinct bony depression of the right vault associated with a weakness of the left arm and left leg, and the history of persistent headaches and of their increasing severity and, during the past year, of several epileptiform seizures in spite of the routine medical treatment, it was therefore considered advisable to perform a right subtemporal decompression in the hope that a lessening of this increased intracranial pressure would cause a definite improvement—even though the injury had been of such long duration.

Operation (17 years after injury).—Right subtemporal decompression: usual vertical incision, bone removed and no complications; the temporal muscle, however, contained a large amount of connective tissue, due undoubtedly to a former hemorrhage in its fibres; the bone at the upper portion of the opening was thickened and vascular, as though a fracture had been present in this area. Dura rather whitish and tense, and upon incising it clear cerebrospinal fluid spurted a distance of one-half inch, and upon enlarging the dural opening a large amount of cerebrospinal fluid escaped, allowing the underlying bulging cortex to recede and pulsate normally. Cortex very "wet" and edematous, with a whitish induration along the vessels in the sulci —the evidence of a former subarachnoid hemorrhage; no gross lesion of the brain ascertained, and as the depressed area of bone above was very slight, it was decided not to remove it in the belief that a simple decompression would suffice. Usual closure with 2 drains of rubber tissue inserted. Duration, 45 minutes.

Post-operative Notes.—The operative recovery was uneventful; within 3 days after operation, the headaches lessened and at the time of discharge—11 days after operation—no convulsions had occurred; the incision healed *per primam.*

Examination (April 10, 1917—15 months after operation).—Patient has improved in every way so that she has been able to work during the past 6 months; only an occasional headache and but 3 convulsions since the operation—the last one being 4 months ago and of a very light character, hardly more than that of *petit mal.* Decompression area slightly depressed and pulsates normally. Definite improvement of use and strength of left arm and left leg. Reflexes—patellar increased, left more than right; no ankle clonus and distinct plantar flexion (normal); abdominal reflexes present and equal. Fundi—retinal veins of normal size; no definite blurring of nasal margins of optic disks but the presence of the new tissue formation naturally persists. Röntgenogram reveals the decompression opening with three silver clips *in situ* (Fig. 133).

Last Report (August 21, 1918—31 months after operation).—Patient writes that she is in good health, works daily, and has no complaints other than a dull headache about once a month, and particularly at the time of her menstruation; has only had one "light spell" during the past 10 months; to her friends, her left arm and left leg are the same as her right arm and right leg in that her gait is normal, although patient says herself, "I know there is a difference between them."

Remarks.—The favorable factor in this case is that, although the cranial injury was of many years' duration, yet the convulsive seizures did not occur until one year before the operation—that is, 16 years after the injury, and at the time of the operation only 7 convulsions had been observed; the prognosis, therefore, for this patient is more hopeful and a complete recovery from the convulsive seizures may be obtained. It would appear that these convulsions had been due to the increased intracranial pressure of a chronic cerebral edema which aggravated the cortical irritation due to the former subarachnoid hemorrhage and, therefore, by simply lessening this increased intracranial pressure by a subtemporal decompression, a marked improvement was obtainable. The bony depression of the vault was of such a slight extent that an ipsolateral decompression was alone necessary. The definite improvement of the left hemiplegia and the early cessation of the headaches are most impressive. It will be necessary, however, to wait for a period of at least 5 years before estimating the permanent benefit of the operation.

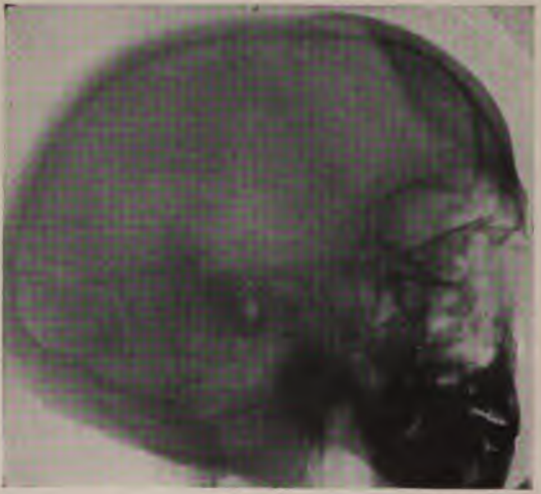

FIG. 133.—Oval bony defect of right subtemporal decompression in a patient having an increased intracranial pressure with persisting symptoms and signs following a cranial injury. Marked improvement. Three silver clips upon the meningeal vessels can be seen in the bony defect.

It is difficult to explain the mild left hemiplegia unless we consider it as having been due originally to a film of supracortical hemorrhage following the cranial injury associated with a depressed fracture of the overlying vault; as this subdural and subarachnoid blood was gradually absorbed by natural means, the left hemiplegia improved until only the mild signs of it remained; as the result of the depressed fracture of the bone overlying the right cerebral motor cortex and the persistence of a chronic cerebral edema, especially of this same area of the brain, the signs of a mild left hemiparesis could still, years later, be elicited by special tests, so that a mere right subtemporal decompression was sufficient in itself to obtain a marked improvement. Naturally, if there had been a lesion of this cerebral cortex sufficient to destroy and impair permanently the cortical nerve cells, then it would have been impossible for an improvement of marked degree to occur in that the cortical nerve cells do not regenerate; fortunately, however, in the majority of patients the intracranial lesion following brain injuries is usually one of compression from hemorrhage and edema to the degree only of functional impairment, and not a destruction or impairment of permanent character.

CASE 107.—Old severe brain injury associated with a depressed fracture of the left parietal area of the vault and with signs of an increased intracranial pressure; right hemiplegia and spells of *petit mal.* Left subtemporal decompression. Improvement.

No. 24.—Hilda. Twenty-one years. White. Single. Clerk. Germany.

Admitted February 4, 1915—19 years after injury. Hospital for the Ruptured and Crippled. Referred by Doctor Virgil P. Gibney.

Operation March 26, 1915. Left subtemporal decompression.

Discharged April 19, 1915—24 days after operation.

Family history negative.

Personal History.—Third child, 9 months' pregnancy, normal labor with no instruments being used; apparently a normal child until the cranial injury. When patient was 18 months of age (19 years ago), she fell from a third-story window, striking upon the left side of her head; immediate paralysis of entire right side of body; remained unconscious for 6 days with profuse bleeding and discharge of cerebrospinal fluid from the left ear; gradually recovered so that the patient apparently developed normally, both mentally and physically, with the exception of a slight stiffness and weakness of the right arm and right leg; no convulsions at any time. Patient left school at the age of 14 years and was able to work as a clerk, always using the left arm in preference to the right arm (both parents, however, and her brothers and sisters were all right-handed). Six months ago, patient noticed that her occasional headaches were increasing in frequency and severity; and at the same time the right hand became weaker and the right leg felt "heavy and stiff," so that a definite lameness of the right leg appeared. Three months ago, she became aware of a slight weakness of the left hand and left arm so that her penmanship became more and more difficult; it was then that her first "fainting spell" occurred while the patient was eating—loss of consciousness of not more than 10 seconds' duration, but sufficient to be observed by the other members of the family. Since this time and during the past 3 months, she has had several lapses of consciousness (*petit mal* attacks), the headaches have increased while the stiffness and weakness of the arms and legs have become more and more marked until she came to the hospital for treatment.

Examination upon admission (19 years after injury).—Temperature, 98.6°; pulse, 84; respiration, 24; blood-pressure, 130. Well-developed and nourished. Over the left parietal bone is a depressed area of almost 3 inches in diameter as ascertained by palpation. Marked right spastic hemiplegia, right arm being affected more than the right leg; only slight right facial weakness—elicited by special tests alone. No sensory impairment. Hearing of left ear not so acute as that of right ear; air conduction, however, is greater than bone conduction in both ears. Mentally—slight retardation and loss of initiative; emotionally of the depressed type, sluggish and "happy go lucky" (as her sister expressed it); since the onset of the attacks of momentary losses of consciousness (spells of *petit mal*), patient has been confused mentally, her memory impaired, especially for recent events, and "in many ways a changed girl." Patient remained in the hospital for 51 days, during which time she was repeatedly examined and her condition carefully observed. Pupils equal and react normally. Reflexes—patellar very much exaggerated, right more than left; inexhaustible right ankle clonus and right Babinski; inconstant left Babinski; abdominal reflexes—right difficult to elicit. Fundi—retinal veins enlarged; nasal margins of both optic disks blurred, left more than right; disk margins themselves rather irregular from new tissue formation. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 16 mm.); Wassermann test negative; cell count was 5 cells per c.mm. X-ray—"over the left parietal area of the vault are two wide lines of fracture with a distinct depression of about 2½ inches in diameter and extending down to the left parietal crest" (Fig. 134).

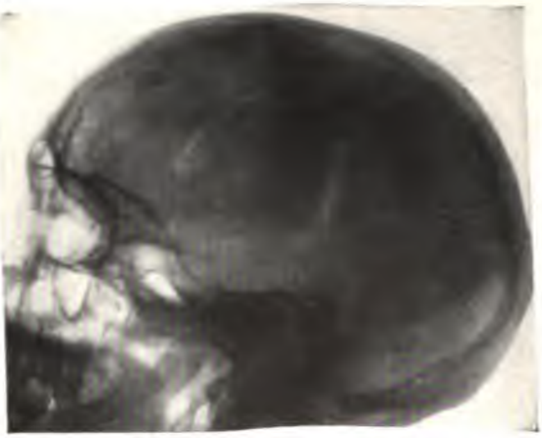

Fig. 134.—Two wide linear fractures of left vault in a patient who had received a cranial injury at eighteen months of age, with a resulting right hemiplegia and epileptiform spells. Definite improvement following a left subtemporal decompression.

Treatment.—The presence of the signs of an increased intracranial pressure associated with a definite right hemiplegia which has become worse even with competent medical treatment, during the past 6 months since the onset of severe headaches and spells of *petit mal,* made a left decompression operation advisable even at this late date following the original brain injury; naturally, it would seem that an operation should have been performed at the time of the injury rather than at this late date.

Operation (19 years after injury).—Left subtemporal decompression: usual vertical incision, bone removed and no complications; bone was unusually thick, almost one cm., and very vascular. Dura thickened, whitish and under high tension; upon incising it, a small amount of cerebrospinal fluid escaped, and upon enlarging dural opening, the underlying cortex appeared almost like liver tissue, in that it was filled with multiple punctate hemorrhages and having a supracortical bluish cystic formation (it would seem that this condition was of shorter duration than our history would i cate—19 years since the cranial injury). Sufficient cerebrospinal escaped, together with the evacuation of the straw-colored fluid o bluish subarachnoid cysts, to permit the cortex to bulge less tensely a pulsate normally at the end of the operation. Usual closure with 2 d of rubber tissue inserted. Duration, 50 minutes.

Post-operative Notes.—Uneventful operative recovery; spasticity of

arm lessened within one week following operation and even on the f
day post-operative, patient was able to extend the fingers of right
"more than ever in my life"; incision healed *per primam*.

Examination at discharge (24 days after operation).—Tempera
98.8°; pulse, 76; respiration, 22; blood-pressure, 132. No headache an
"heavy feeling" in the head has almost entirely disappeared. M
improvement of the spasticity of the right arm and the right leg. No
of *petit mal* type since operation. Decompression area flush with the
rounding scalp. Reflexes—patellar exaggerated, right more than
inexhaustible right ankle clonus and right Babinski still persist; ab
inal reflexes—right less active than left. Fundi—retinal veins enla
nasal margins more distinct than before operation, though the newly fo
tissue about the optic margins naturally is present.

Examination (January 10, 1917—23 months after operation and 21
after injury).—Patient has made a marked improvement: right hemip
has lessened though it is still present; an occasional dull headache
the spells of *petit mal* have only occurred 9 times during the past 2 y
She is able to work daily and is now using her right hand to per
simple acts; she has more "ambition" according to sister. Decom
sion area slightly depressed. Reflexes—patellar exaggerated, right
than left; inexhaustible right ankle clonus and right Babinski; ab
inal reflexes—right less active than left. Fundi—retinal veins sli
enlarged; nasal margins, however, of both optic disks distinct though ir
lar from newly formed tissue.

Last Report (October 10, 1918—43 months after operation).—
writes that patient is now living out West and that her condition ha
improved since last examination one year ago; she had, however,
3 spells of *petit mal* during the past year and was "sufficiently well"
married 2 months ago. She no longer complains of headaches and "
to be enjoying life."

Remarks.—The pathology as revealed at operation was of such an e
sive character and in the cortex itself that the ultimate prognosis mu
considered very grave, even in spite of this definite improvement follo
operation. Naturally, the chief danger lies in the formation of the so-
epileptic "habit" due to the cortical irritation as the result of the co
hemorrhage; this process undoubtedly has been retarded by the less
of the increased intracranial pressure by means of the decompression,
seems too much to expect an ultimate cure to be effected.

There must have been in this patient a destruction of some of the co
nerve cells due to the multiple punctate hemorrhages among them, but
other of the cortical nerve cells had been merely compressed by these
tate hemorrhages and the resulting cerebral edema, so that when th
creased intracranial pressure was finally lessened by means of the decom
sion (even at the late date of years following the original injury),
compressed cells functionally were now enabled to regain their former
ity to a greater or less degree and thus an almost immediate
the patient's condition appeared. Naturally

intracranial pressure by means of the decompression should have been performed years ago—better at the time of the original injury.

It is surprising how well children under 2 years of age and even older, apparently recover following severe cranial injuries; unfortunately, however, it does happen rather frequently that a child entirely recovers only clinically, and if more careful and thorough examinations were made at the time of the injury and over a period of weeks and even months following the injury, it could be very easily ascertained whether there were intracranial signs present indicative of future complications—particularly the presence or not of an increased intracranial hemorrhage; in those children in whom the increased intracranial pressure is high following the injury, although they apparently make an excellent recovery from the acute condition, yet it is in these children that future complications occur, such as mental retardation and emotional instability and epileptiform seizures of major and minor character. Their history is so frequently that of being considered normal children, even until the age of puberty when the cortical nerve cells develop qualitatively, as it were, and it is then that the definite signs of impairment appear, and so frequently associated with convulsive seizures of varying degree. All children having severe cranial injuries should be considered as being seriously injured until repeated thorough examinations, particularly with the ophthalmoscope and the measurement of the cerebrospinal fluid at lumbar puncture, as registered by means of the spinal mercurial manometer; naturally, exhaustive neurological examinations are essential.

Case 108.—Old severe brain injury associated with a depressed gunshot fracture of the vault and with signs of an increased intracranial pressure; removal of depressed bone alone and insertion of silver plate; convulsive seizures. Right subtemporal decompression and removal of silver plate. Improvement.

No. 1047.—Paul. Eighteen years. White. Single. Soldier. Russia.

Admitted November 24, 1918—39 months after injury. Audubon Hospital. Referred by Doctor James A. Harrar.

Operation December 5, 1918—11 days after admission. Right subtemporal decompression.

Discharged December 24, 1918—19 days after operation.

Family history negative.

Personal History.—Always well and strong until the present cranial injury. On August 28, 1915 (39 months ago), while fighting as a private soldier in the Russian army in Galicia, patient was struck over the posterior portion of the right fronto-parietal area near the midline by a shell fragment, producing a depression of the underlying bone; he was found unconscious and taken to an advanced emergency hospital, where the depressed area of bone was removed and a metal plate inserted beneath the scalp within 24 hours after the injury; he remained unconscious for 3 days, having a paralysis of the entire left side of the body, which began to improve, however, 6 days after operation; he remained in the hospital 4 weeeks, but the wound did not heal until 14 months later, owing to a purulent discharge from the posterior portion. In October, 1915 (2 months after injury), patient had his first general convulsive seizure—no localizing signs being observed; in December, 1915 (4 months after injury), the second general convulsive seizure occurred and at this time it was noted that the left arm and left leg convulsed first, and then the right side of the body was involved; patient complained of continuous dull headache. The third convulsion was in May, 1916 (9 months after injury), and the last convulsive seizure occurred in September, 1918 (2 months ago), following a bicycle-ride; no localizing signs were observed. During these past 3 years since the injury, patient has complained almost daily of dull headache. He has, however, improved in every way—there being no gross sign present of the former left hemiplegia. Father states that the patient is not so alert mentally as formerly, and is emotionally unstable—flying into fits of anger upon slight provocation.

Examination upon admission (39 months after injury).—Temperature, 98.8°; pulse, 78; respiration, 22; blood-pressure, 134. Unusually well-developed and nourished. Patient spoke only Russian, so that it was impossible to elicit the mental retardation as stated by the father; patient was very restless, however, and very irritable as illustrated by his throwing a glass of drinking water at a nurse for not being more attentive to him. Over the posterior portion of the right parietal area was a curvilinear scar of 4 inches in length; the underlying bone was irregular but no marked depression palpable. No paralysis nor sensory impairments. Hearing negative; otoscopic examination negative. Pupils equal and react normally. Reflexes—patellar exaggerated, left possibly more active than right; no ankle clonus but suggestive left Babinski; abdominal reflexes present and equal. Fundi—retinal veins enlarged; nasal margins of both optic disks blurred by edema; physiological cups both shallow from new tissue formation. Lumbar puncture—clear cerebrospinal fluid under increased pressure (16 mm.); Wassermann examination negative and cell count was 5 cells per c.mm. Urine examination negative. X-ray (Doctor A. J. Quimby)—"dense circular shadow over the right fronto-parietal area of the size of 3 inches in diameter—most probably a silver plate covering an underlying bony defect" (Fig. 135).

Treatment.—The history of the cranial injury with subsequent convulsive seizures—the last one being 2 months ago, and associated with definite signs of an increased intracranial pressure with mental and emotional impairment, these facts make advisable a lessening of this increased intracranial pressure by means of a right subtemporal decompression in the hope that the condition will be improved; also the irritation of the foreign body over the right cerebral cortex should be removed. This patient has been under competent medical treatment during the past 2 years at least.

Operation (39 months after injury).—Right subtemporal decompression: usual incision, bone removed and no complications; an unusual amount of bleeding owing to the vascularity of the scalp, muscle and of the bone itself; a branch of the right middle meningeal artery was torn by the rongeurs so that it required ligation by means of a silver clip. Dura tense, opaque, thickened and very vascular; upon incising it, clear cerebrospinal fluid spurted to a height of 3 inches, and upon enlarging the dural opening, a large bluish cystic mass was exposed, lying in and upon the underlying cortex —the cystic residue of a former supracortical and cortical hemorrhage;

large dilated cortical vessels passed through it, and upon incising it carefully, a straw-colored fluid welled out to the amount of almost 1 ounce. So much cerebrospinal fluid escaped that the intradural tension was lessened and the cerebral cortex receded and pulsated almost normally. Much fibrous induration in the arachnoid, and particularly about the cortical vessels in the sulci—the residue of a former subarachnoid hemorrhage. Vertical incision now enlarged upward and the silver plate easily removed; many dural adhesions to it. Usual closure with 2 drains of rubber tissue inserted. Duration, 60 minutes.

Post-operative Notes.—Uneventful operative recovery; the dull heavy feeling of the head lessened and at the end of one week, the patient told his father that his headache had gone; patient was discharged on the sixteenth day after operation; incision healed *per primam;* operative area pulsates normally.

Last Report (September 12, 1919—10 months after operation).—Father states that the patient has made a marked improvement in that no convulsive seizures have occurred, the headaches have entirely disappeared and the patient is more stable emotionally; he is studying engineering in England and has become very much interested in his work; "it is now possible for him to learn, whereas before he could not concentrate his mind upon anything." Operative area does not bulge beyond the flush of scalp.

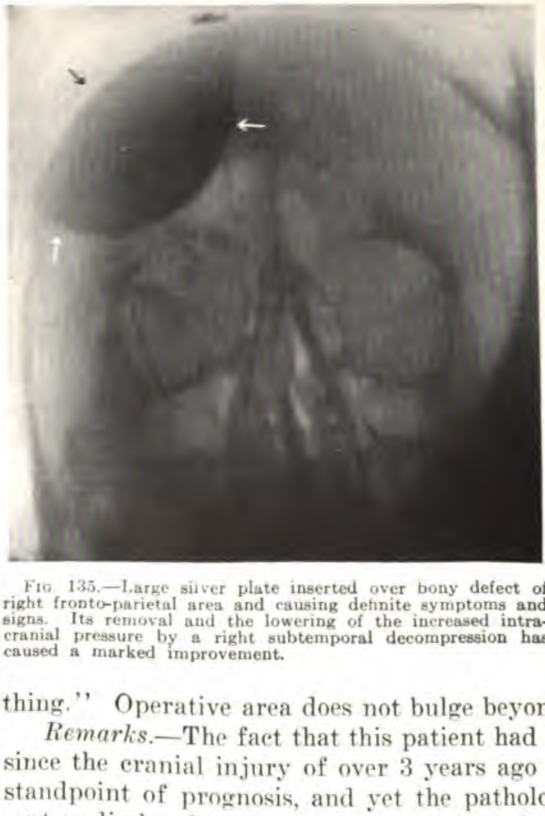

FIG. 135.—Large silver plate inserted over bony defect of right fronto-parietal area and causing definite symptoms and signs. Its removal and the lowering of the increased intracranial pressure by a right subtemporal decompression has caused a marked improvement.

Remarks.—The fact that this patient had had only 4 convulsive seizures since the cranial injury of over 3 years ago is very encouraging from the standpoint of prognosis, and yet the pathological lesion of a hemorrhagic cyst as disclosed at the operation and its being in the cortex and not merely upon the cortex, as is usually the case, makes the ultimate recovery from the convulsions very doubtful; the removal of the silver plate, however, may be very beneficial. It is most unfortunate that this operative procedure of decompression and drainage was not performed within a short time following the injury rather than at this late date, when only the pressure effects of this hemorrhage and the resulting cerebral edema can be lessened and offset, whereas following the injury upon the patient's recovery from the shock, the operation of subtemporal decompression and drainage would have drained off the hemorrhage itself and thus an excellent result have been obtained and no convulsive seizures probable! Merely elevating or removing the depressed bone of the vault (besides the insertion of foreign bodies for "protection") and not relieving the increased intracranial pressure when associated with intradural hemorrhage and marked cerebral edema,—this method of treatment is not sufficient and these traumatic sequelæ of depressed fractures of the vault are only too common following the local operation upon the depressed area of bone alone.

The supracortical hemorrhage must have been very large and extensive to have produced a complete left hemiplegia; its gradual absorption, however, so that the hemiplegia has practically disappeared, would indicate that the pathology as disclosed at the operation may now be limited to this comparatively silent area of the right temporo-sphenoidal lobe, and naturally the clinical signs of its presence cannot be elicited—other than the signs of a general increase of the intracranial pressure and the slight neurological changes. Naturally, it will be necessary for a number of years to elapse before an opinion regarding this patient's ultimate recovery can be given.

c. *Two operations: decompression first, then removal of the depressed area of vault—there being present definite signs of a marked increase of the intracranial pressure.*

Case 109.—Old severe brain injury associated with a depressed fracture of the vault and with signs of an increased intracranial pressure; convulsive seizures. Two operations: left subtemporal decompression first, and then a removal of the depressed area of bone. Improvement.

No. 115.—Joseph. Fourteen years. White. School. U. S.

Admitted March 1, 1914—7 years after injury. Polyclinic Hospital. Referred by Doctor M. Allen Starr.

Operations April 2, 1914. Left subtemporal decompression and removal of depressed area of vault.

Discharged April 10, 1914—8 days after operations.

Family history negative.

Personal History.—Always well and strong until cranial injury. Seven years ago, when patient was 7 years of age, he was kicked by a horse over the posterior portion of the left frontal area; no loss of consciousness; patient was taken to a hospital in the ambulance and a "bone pressing on the brain was removed"; upon his discharge from the hospital 3 months later, it was noticed that the right leg was much weaker than the left and that its movements were more awkward; also "his mind seemed to be in a cloud." This condition continued until 2 years ago (patient then being 12 years of age), when the first general convulsive seizure occurred—no localizing signs being observed; he had been complaining of headache which now became much worse. Four weeks later, the second general convulsive seizure occurred, to be followed by similar convulsions with increasing frequency and severity until it was necessary, 7 months ago, to place the patient in an institution. During these past 7 months in the institution, he has complained of persistent headaches, convulsive seizures have occurred almost daily and the personality of the patient has entirely changed, in that now he has sudden fits of anger associated with wanton cruelty—being a danger and menace to other children, even necessitating his isolation and restraint at times.

Examination upon admission (7 years after injury).—Temperature, 98.6°; pulse, 80; respiration, 22; blood-pressure, 124. Well-developed and nourished. Over the posterior portion of left frontal bone is a depressed area of 1½ inches in diameter; no pulsation palpable. Patient seems confused mentally, retarded and in a very irritable condition. Hearing negative. Pupils equal and react normally. Reflexes: patellar—right more active than left; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi—retinal veins dilated; nasal halves of both optic disks obscured by edema; much new tissue formation about optic disk margins and in physiological cups, making both optic disks rather pale and thus giving the appearance of a mild secondard optic atrophy. Lumbar puncture—clear cerebrospinal fluid under increased intracranial pressure (approximately 16 mm.); Wassermann test negative. X-ray (Doctor A. J. Quimby)—"indistinct bony defect of 2 cm. in diameter in posterior portion of left frontal bone; surrounding bony edge also depressed."

Treatment.—The definite signs of an increased intracranial pressure and the presence of the bony defect and depression of the surrounding bone, together with the increased reflexes upon the right side—and especially after a competent and thorough medical treatment had failed to cause an improvement of the convulsive seizures, these facts made a left subtemporal decompression advisable in the hope that the condition might be benefited; naturally, if a decompression operation at this late date could improve the condition of the patient, how much more benefit would an earlier operation have obtained—possibly the convulsive seizures might even have been prevented from occurring at all.

Operations (7 years after injury).—First, left subtemporal decompression: usual vertical incision, bone removed, and no complications. Dura was thickened and under moderate tension; upon incising it, much cerebrospinal fluid escaped, revealing a very "wet," edematous cortex; arachnoid was of cloudy appearance and about the vessels in the sulci was a whitish induration—the residue of a former subarachnoid hemorrage. Occasional adhesions found between the arachnoid and the overlying dura. Brain pulsated at end of operation. Usual closure with 2 drains of rubber tissue inserted. Temporary sterile dressing applied.

Second Operation.—Curvilinear incision of 2 inches over depressed area of left frontal bone; small trephine opening made at posterior edge of bony depression and the depressed area of spongy bone removed—new bone formation apparently. Dura was thickened and vascular, requiring the application of three silver clips; upon incising it, there was revealed an underlying cystic formation, bluish in color, and at least one-half inch in thickness; this cyst was punctured, allowing a straw-colored fluid to escape. Dura closed with silk. Usual closure of scalp with one drain of rubber tissue inserted. Duration, 65 minutes.

Post-operative Notes.—Uneventful operative recovery; at discharge, patient no longer complained of headache and did not appear so irritable. No convulsions have occurred since the operation; incision healed *per pri-*

mam. A second X-ray picture shows "the two bony defects of the left vault; also three silver clips within a decompression area" (Fig. 136).

Examination (June 12, 1914—70 days after operation).—No complaints in that the headaches have ceased and no convulsions have occurred; from reports of his teachers in school, he is "a changed boy—not so unruly and has better control of his temper; is more attentive to his studies." Decompression area slightly depressed and pulsates normally. Reflexes—patellar active but equal; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi—retinal veins only slightly enlarged; no blurring edema of the nasal halves of the optic disks but the mild signs of a secondary optic atrophy naturally persist.

Examination (April 6, 1916—24 months after operation).—Since the operation, patient has had 9 convulsions—the first one being 9 months after the operation; patient does not complain of headaches and he has made a marked improvement in his school work; he is emotionally more stable and no longer "loses his temper." Decompression area slightly depressed. Reflexes active but otherwise negative. Fundi—retinal veins slightly enlarged; no edematous blurring of either optic disk; the mild secondary optic atrophy is present.

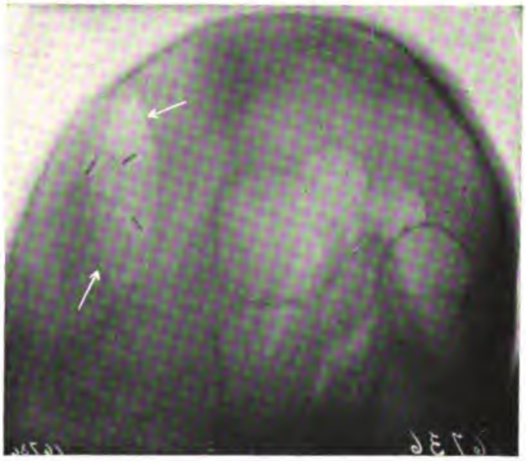

FIG. 136.—Oval bony defect of left subtemporal decompression in a patient having (seven years ago) a depressed fracture of the left vault followed by convulsive seizures. Marked improvement following the operative lowering of the increased intracranial pressure.

Last Report (July 10, 1918—52 months after operation).—Mother writes that "Joseph has had 11 convulsions during the past year but they do not upset him as they did before the operation; he no longer has headache and seems bright in every way."

Remarks.—Although this patient has undoubtedly been benefited by the operation in relieving the increased intracranial pressure and thereby lessening the cortical irritation, so that the convulsive seizures have been of less frequency and severity, yet the ultimate result, I feel, will be merely that the condition has been delayed and that the usual mental deterioration and emotional instability will result finally, as in practically all of these patients having convulsive seizures; the operation, however, was surely justified and if this patient had received the appropriate treatment immediately following the injury, it might have been possible to have prevented these convulsive seizures from occurring at all—at least, the patient would have had a greater chance of approximating a normal individual. The operative findings of a former subarachnoid hemorrhage and a definite hemorrhagic cystic formation immediately beneath the bony depression of the vault and lying directly upon the cortex—these findings in themselves are undoubtedly of sufficient cortical irritation to produce the convulsive seizures, even in the absence of an increased intracranial pressure and especially after the cortex itself has become irritable as the result of numerous preceding convulsions appearing over a period of several months and even years. It will be interesting to follow this patient during the next 5 and 10 years. The following note was made within 2 months after his operation: "Considering the cortex as ascertained at operation, it seems incredible that the boy can remain improved permanently; yet in adolescents, it appears that the cortex can return to a stable and less irritable condition of abnormality more easily than in adults, even though the traumatic epilepsy has persisted over a number of years."

CASE 110.—Old severe brain injury associated with a depressed fracture of the right frontal bone and with signs of an increased intracranial pressure; spells if *petit mal* and severe headache. Two operations: right subtemporal decompression and then a removal of the depressed area of the vault. Recovery.

No. 130.—Arthur. Thirty-seven years. White. Married. Coal-miner. United States.

Admitted April 13, 1914—4 years after injury. Polyclinic Hospital. Referred by Doctor John I. Van Wert, Patton, Pa.

Operations (April 21, 1914—4 years after injury).—Right subtemporal decompression and removal of depressed area of vault.

Discharged May 3, 1914—12 days after operations.

Family history negative.

Personal History.—Always well and strong. Four years ago, patient was struck over the right forehead by a wooden plank in a mine explosion; loss of consciousness for several minutes; laceration of scalp over the right forehead but no depression of the underlying bone ascertained; patient remained at home and in bed for 17 days, having severe headache and dizziness. Four months later, patient attempted to work but was unable to work longer than one day on account of the severe right frontal headache and dizziness; during the past 3 years he has been able to work 1 or 2 days at a time and then he had to remain at home for a period of one week to one month on account of the severe headache, dizziness and, during the past 6 months, momentary losses of consciousness while eating, talking and when he worked. Three years ago, the right frontal sinus was exposed and curetted but no real improvement resulted. Beside the headache, dizziness and the spells of *petit mal*, patient had become mentally and emotionally depressed, slept poorly and complained of a blurring of vision. Hi
dition had been diagnosed at several clinics as being one of traumatic r
thenia and as hysteria.

Examination upon admission (4 years after injury).—Temp
98.8°; pulse, 62; respiration, 20; blood-pressure, 160. Fairly well-de
and nourished. A slight depression of the right frontal bone of 2 i
width and 1 inch in length; marked local tenderness. Pupils

react normally. Reflexes—patellar present and equal; no ankle clonus nor Babinski; abdominal reflexes—left possibly less active than right. Fundi—retinal veins dilated; edematous blurring of nasal margins of both optic disks, which were rather pale due to new tissue formation and thus producing a mild secondary optic atrophy. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 16 mm.); Wassermann test negative and cell count was 4 cells per c.mm. X-ray (Doctor A. J. Quimby)—"in right frontal area, two inches above right supra-orbital ridge, is an irregular bony depression and defect of the right frontal bone just to the right of the midline; it is rather hazy and indistinct at this point; no linear fracture shown" (Fig. 137).

Treatment.—If it were not for the presence of the signs of increased intracranial pressure, it would be very easy to consider this patient as having merely a functional condition with possibly a slight injury to the right frontal bone itself; the signs of an increased intracranial pressure, however, immediately cause this patient to be removed from that large group of patients properly classified as functional and to be placed among those patients having definite organic conditions intracranially as the result of the cranial injury. It was thought advisable to perform a subtemporal decompression to lessen the increased intracranial pressure and then to remove the depressed area of bone in the belief that it might be a source of cortical irritation.

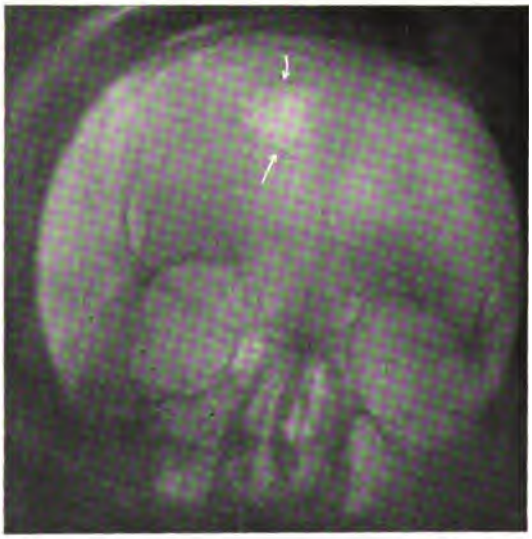

FIG. 137.—Old depressed fracture of the right frontal bone followed by severe continuous headache and epileptiform spells, and associated with an increased intracranial pressure. At operation, a piece of wood (4 x 2 cm.) was found in the depressed area of bone.

Operations (4 years after injury).—First, right subtemporal decompression: usual vertical incision, bone removed, and no complications; bone was unusually thick with several large sinuses in the diploë. Dura was thickened and under marked tension; upon incising it, much cerebrospinal fluid escaped, revealing a very "wet," edematous cortex; the arachnoid was "smoky" and about the vessels in the sulci was much connective-tissue formation—the residue of a former subarachnoid hemorrhage; there were many adhesions between the arachnoid and the overlying dura. The cortex bulged so that much cerebrospinal fluid escaped and the arachnoid "sweated" so profusely that the cortex receded and pulsated normally at the end of the operation. Usual closure with 2 drains of rubber tissue inserted. Temporary gauze dressing applied.

Second Operation.—Removal of depressed area of right frontal bone and extraction of the foreign body; curvilinear incision of 2 inches made over the depressed area; healed line of fracture with callus formation was revealed, extending from the area of depression to the left over and beyond the longitudinal sinus, where it bifurcated; protruding from this old line of fracture in the depressed area was a piece of wood 1½ inches long and a quarter of an inch in diameter. The depressed area of bone and the foreign body rongeured away; the underlying dura had apparently not been damaged and therefore it was not opened. Usual closure with 2 drains of rubber tissue inserted. Duration, 70 minutes.

Post-operative Notes. -Uneventful operative recovery; both scalp incisions healed *per primam* and at discharge patient no longer complained of severe headache or of dizzy spells.

Examination (September 20, 1915—17 months after operation).—Patient states that he is better than at any time following the injury; in fact, he no longer has headaches or dizzy spells, not a single attack of *petit mal* has occurred and in every way he seems to be, as he says, "a new man." Patient went to work one month after the operation and has not missed a day since. Decompression area is flush with the surrounding scalp; pulsates normally. Reflexes active but otherwise negative. Fundi—retinal veins slightly enlarged; no blurring of optic disk margins but the mild secondary optic atrophy naturally persists.

Last Report (October 22, 1917—42 months after operation).—A letter has just been received from Doctor Van Wert stating that "the patient was killed one week ago in a mine explosion. The patient had enjoyed excellent health since the operation, had worked daily and there had been no complaints." The tragic ending of this patient is most unfortunate.

Remarks.—It is undoubtedly a rather common mistake in diagnosis to consider post-traumatic conditions in patients as being practically all functional, and therefore frequently no careful neurological examination is made—especially the ophthalmoscopic examination of the fundi and the accurate estimation of the intracranial pressure by means of the spinal mercurial manometer at lumbar puncture; if such patients have an increased intracranial pressure, then they can no longer be considered as purely functional, and their post-traumatic symptoms of headache, vertigo, loss of sleep, emotional instability and the other numerous so-called neurasthenic symptoms and signs, when associated with an increased intracranial pressure, have thus a definite organic basis for their impairment and should be treated accordingly; post-traumatic neurasthenia and hysteria have been too frequently applied to post-traumatic conditions merely because, in many patients, careful examinations were not made.

It was rather surprising in this patient to find the piece of wood iml
in the bone: it in itself, however, was not the cause of the patient's cor
but rather the "wet," edematous condition of the brain resulting f
former subarachnoid hemorrhage and its resulting cerebral edema.

Case 111.—Old severe brain injury associated with a depressed
of left parietal area of vault and with signs of increased intr
sure: paraphasia and convulsive seizures. T

decompression and then a removal of the depressed silver plate covering the bony defect. Excellent recovery.

No. 180.—John. 37 years. White. Married. Hostler. United States.

Admitted March 15, 1915—10 months after injury. Polyclinic Hospital. Referred by Doctor J. R. Bingham.

Operations (March 27, 1915—10 months after injury).—Left subtemporal decompression and removal of depressed area of vault.

Discharged April 12, 1915—16 days after operations.

Family history negative.

Personal History.—Patient has always been well and strong; never any severe illness; his life has been rather a hard, rough one and associated with much drinking. Ten months ago (June, 1914), patient was kicked upon the left side of head by a mule; loss of consciousness for 30 minutes; the scalp laceration was sutured and, upon recovering consciousness, the patient was unable to speak; this condition of aphasia lasted for 5 weeks and has never entirely disappeared. Six weeks after the injury, patient had his first general convulsion and these continued at intervals of 2 weeks. Six months ago (October, 1914), a left osteoplastic bone flap operation was performed at a hospital in Baltimore—the bone flap being removed at the end of the operation and a large silver plate placed over the bony defect. A definite improvement of the aphasia followed but the convulsive seizures returned after a period of 8 days with even greater severity than before the operation; since then, the "fits" have occurred every 6 to 10 days. Six weeks ago while aboard ship at a port in France, patient became intoxicated and during a fight was struck over the head by a heavy bottle; immediate loss of consciousness for 2 hours and since that time, the convulsive seizures have become of still greater severity and frequency—one every 4 days and lasting 2 to 3 hours each time.

Present Illness.—Six hours ago, while standing upon the street corner, the patient had a severe convulsion, general in character and lasting 2 hours; unable to talk during the following 3 hours; brought to the hospital in the ambulance.

Examination upon admission (10 months after the original cranial injury).—Temperature, 99°; pulse, 76; respiration, 18; blood-pressure, 142. Drowsy and confused mentally; complains of severe headache. Well-developed and nourished. Over the left parietal area of the vault was the curvilinear scar of the former operative incision arching over the earlier scalp wound; the silver bone plate was depressed to a depth of almost 1 cm. Hearing negative. Definite paraphasia; the speech was also very thick. Pupils equal and react normally. Reflexes: patellar, active but equal; no ankle clonus but suggestive right Babinski; abdominal reflexes, both depressed—right possibly more than left. Fundi: retinal veins full; nasal margins of both optic disks blurred by edema. Lumbar puncture: clear cerebrospinal fluid under increased pressure (approximately 15 mm.); Wassermann test, negative and cell count was 7 cells per c.mm. X-ray (Doctor A. J. Quimby)—"silver plate of former operation had become depressed to a depth of almost ½ inch; no linear fracture ascertained" (Fig. 138).

Treatment.—Patient was carefully examined during a period of 10 days;

at the end of that time as the signs of an increased intracranial pressure persisted and the patient had had 3 convulsions of a general character, although the right arm each time was involved before the left side of the body, it was considered advisable to perform a left subtemporal decompression first and then to remove the depressed silver plate.

Operations (10 months after injury).—First, left subtemporal decompression: usual vertical incision, bone removed and no complications; rather anterior incision in order to expose the motor speech area (the patient and his relatives all being right handed); at the upper angle of the bony opening, there was a narrow bridge of bone, ½ inch in width, between the decompression opening and the line of fracture of the former osteoplastic operation. Dura was thickened, whitish and under tension; upon incising it, clear cerebrospinal fluid oozed out, revealing a very "wet" edematous cortex; about the vessel walls in the sulci and in the arachnoid itself was a whitish induration—the residue of a former subarachnoid hemorrhage. A large supracortical vein was accidentally punctured, but its bleeding was quickly controlled by the application of a small piece of temporal muscle (which hastens the formation of a clot and thus facilitates the hemostasis). Owing to the escape of much cerebrospinal fluid, the cortex now became relaxed and pulsated normally. Usual closure with 2 drains of rubber tissue inserted. Temporary gauze dressing applied.

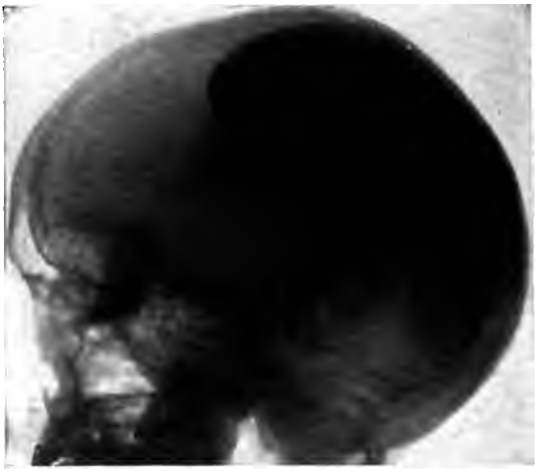

FIG. 138.—Large oval silver plate used to cover a bony defect of the left parietal area. The silver plate became depressed, causing persisting symptoms and signs; its removal and a left subtemporal decompression permitted an excellent recovery.

Second Operation.—Curvilinear incision of 2 inches over the center of the depressed silver plate; small trephine opening made, enlarged by rongeurs to a diameter of 2 inches, and thus it was possible to remove the depressed silver plate. The underlying dura was thickened but not under tension, and therefore it was not opened. Usual closure with 2 drains of rubber tissue inserted. Duration, 80 minutes. Post-operative notes: Except for extreme restlessness, the patient made an uneventful operative recovery; no convulsions occurred during his convalescence and as the headache had disappeared, the patient insisted upon his discharge on the eleventh day.

Examination (February 20, 1917—25 months after operations).—Patient has had only 4 convulsions since the operation—the last being 11 months ago; he has been at work daily on an ice-wagon and insists that he is no longer drinking—a very important factor in this case. The decompression area is flush with the surrounding scalp, while the scalp is rather sunken at the site of the craniectomy and removal of the silver plate. There is a definite improvement of speech and the patient's entire general condition has been benefited. Reflexes active but otherwise negative. Fundi: retinal veins slightly enlarged; optic disks clear but nasal margins are rather irregular from new tissue formation.

Last Examination (August 28, 1918—41 months after operation).—Patient was met upon the street delivering ice; "feeling fine." No convulsions since the last examination—that is, during the last 29 months; has an occasional headache but only "light ones"; no longer drinks, and his son of 12 years of age, who was with him, corroborates this statement. Decompression area slightly depressed and pulsates normally. Reflexes active but otherwise negative. Fundi: retinal veins of normal size; optic disks clear while the new tissue formation is still present along their nasal margins.

Remarks.—I believe that alcohol was a definite factor in this man's condition; that is, in addition to the cortical irritation due to the former subarachnoid hemorrhage and the depressed silver plate, that the drinking daily of a large amount of alcohol increased the resulting cortical irritability, and thus not only predisposed the patient to convulsive seizures but also made the patient more susceptible to the so-called "epileptic habit." The lessening of the increased intracranial pressure by the decompression operation and the removal of the depressed silver plate—and at the same time, the cessation of the drinking of alcohol, made it possible for this patient to make an excellent recovery and that both were necessary in order to obtain this good result.

In the presence of an increased intracranial pressure due to a former cranial injury in these patients, the mere osteoplastic operation with an opening of the dura, and then the dura resutured and the bone flap replaced—this operative procedure is not of permanent value in that the increased intracranial pressure is only lessened temporarily, and in these patients having a chronic edematous condition of the brain, it is essential that the decompression and drainage should be a permanent one—as is afforded by the subtemporal decompression; therefore, the subtemporal decompression is the more advisable, but if an osteoplastic flap operation is performed, then it should be associated with a subtemporal decompression to be performed at the same time.

To remove a large portion of the vault of the skull and then to insert a so-called "protecting plate" of silver or other foreign body is distinctly a dangerous procedure and frequently associated with later complications—as in this patient. If a large bony defect is present and of a diameter of more than four inches, then it is better judgment for the patient to wear a tight-fitting skull cap with a metal plate in the cap and covering the bony defect rather than the surgeon attempting to insert beneath the scalp a foreign body of metal and trust that no complications will occur—both the immediate danger of infection and the later dangers of resulting cortical irritation. The inserted foreign body need not be depressed as in this patient in order to produce intracranial complication.

Case 112.—Old severe brain injury associated with a depressed fracture of the left frontal bone and with signs of increased intracranial pressure;

convulsive seizures. Two operations: left subtemporal decompression and then a removal of depressed area of bone. Improvement.

No. 877.—Daniel. Thirty-six years. White. Single. Steward. Ireland.

Admitted June 23, 1917—34 years after injury, Polyclinic Hospital. Referred by Doctor George W. Jacoby.

Operations June 31, 1917. Left subtemporal decompression and removal of depressed area of bone.

Discharged July 17, 1917—17 days after operation.

Family history negative.

Personal History.—Third child, full-term baby, normal delivery; considered a normal child until cranial injury. When 2 years of age (34 years ago), the patient fell a distance of 10 feet, striking left forehead against a curbstone and causing a depression of the left frontal bone; 2 days later, he was operated upon and "a piece of the depressed bone removed, turned around and then placed back"; patient apparently made an excellent recovery and was considered a normal child until 9 years ago, at 27 years of age, when he was first troubled by nausea and dizziness, and finally a general convulsive seizure occurred; within 6 months, these convulsive seizures increased in severity and frequency until they were occurring as often as once in every 10 days to 2 weeks. Eight years ago (February, 1909), patient was operated upon at the German Hospital: "the skull was opened in the frontal region, the bone-flap turned down and the dura exposed; the dura showed an old scar and upon incision pronounced adhesions between it and the brain. A puncture into the frontal lobe resulted negatively. The adherent dura was excised, a silver leaf placed over the cortex and the bone put back. After the operation (and until June, 1912), this patient was carefully watched, but the operation had no effect whatsoever upon his convulsions." During the past 5 years, the condition of the patient has remained practically the same—general convulsive seizures occurring one or more times weekly, and during the past 6 months, he has had one convulsion at least each day; there has been a marked mental and emotional deterioration during this period—there being a marked impairment of memory; continuous severe headaches daily, especially during the past 6 weeks.

Examination upon admission (34 years after injury).—Temperature, 98.6°; pulse, 70; respiration, 20; blood-pressure, 136. Fairly well-nourished and developed. Curvilinear scar over left frontal bone—incision of former operation; depressed area—apparently of bone and of one inch in diameter; hard upon palpation and no pulsation obtained. No paralyses or impairment of sensation. Pupils equal and react normally. Reflexes: patellar—both active, right possibly more than left; no ankle clonus but a suggestive right Babinski; abdominal reflexes—right less active than left. Fundi—retinal veins full; nasal margins and lower nasal quadrants of both optic disks blurred by edema; small amount of new tissue formation along the margins of optic disks and in both physiological cups. Lumbar puncture—clear cerebrospinal fluid under increased pressure (17 mm.); Wassermann test negative and cell count was 8 cells per c.mm. X-ray (Doctor G. W.

Welton)—"depressed area of new bone formation in left frontal area; no linear fracture shown."

Treatment.—Although the prognosis for this patient was practically hopeless, with or without operation—the convulsions having persisted over such a long period of time and following an injury of over 30 years ago, yet as there were present the signs of an increased intracranial pressure as revealed by the ophthalmoscope and as registered by the spinal mercurial manometer, it was considered advisable to perform a left subtemporal decompression and a removal of the new bone formation at the site of the depression in the hope that the cortical irritation might thus be lessened and the convulsive seizures and their resulting mental and emotional deterioration be at least retarded and delayed.

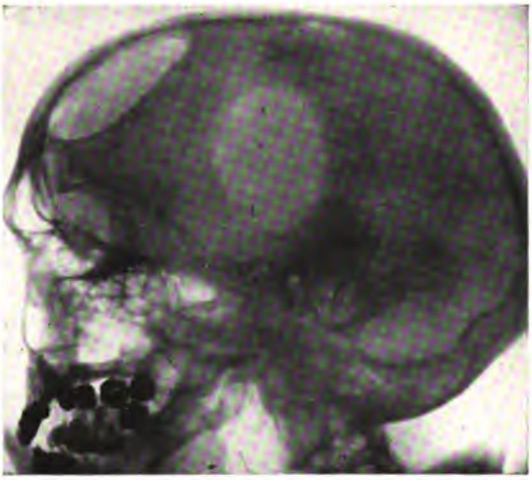

FIG. 139.—The large oval defect of the left frontal bone, following a removal of an old depressed fracture and a left subtemporal decompression in a patient having a high intracranial pressure with convulsive seizures. Marked improvement following the operation.

Operations (34 years after original brain injury).—First, left subtemporal decompression: usual vertical incision, bone removed, and no complications. Dura very thick, vascular and tense, and upon incising it, much clear cerebrospinal fluid escaped, revealing a very "wet," edematous cortex, which protruded under moderate tension but did not rupture. Whitish induration about the vessels in the sulci and of the overlying arachnoid—the possible residue of the former subarachnoid hemorrhage. Usual closure with 2 drains of rubber tissue inserted. Temporary sterile gauze dressing applied.

Second Operation.—Curvilinear incision made through scar of former operation; after much difficulty, the silver plate imbedded in much new bone formation was removed and the numerous adhesions to underlying dura which had apparently re-formed were separated. Upon incising the dura, there was exposed, lying within the cortex itself, a bluish cystic mass—the size of a lemon; the outer wall of the cyst was excised, allowing straw-colored fluid to escape and then the cyst itself collapsed; several adhesions between the cyst-wall and overlying dura were severed. The dural opening and the wound were closed in the usual manner with 2 drains of rubber tissue inserted beneath the scalp. Duration, 80 minutes.

Post-operative Notes.—Uneventful operative recovery; patient, however, had one convulsion during his hospital convalescence; patient seemed less confused mentally and felt that his headaches were less in severity upon discharge—17 days after operation; incision healed *per primam*.

Examination (November 20, 1917—5 months after operations).—After leaving the hospital, patient had his first general convulsion 7 weeks later, to be followed by the second one after a period of 8 weeks; he has had in all now since the operation 5 convulsions and is feeling better than "for years"; he has a "light" position as a watchman—the first time he has been able to earn any money for 9 years. He no longer has headaches, and although his mentality is definitely retarded, he can now remember simple things, and he has sufficient emotional control so that he can live with his relatives and his relatives can live with him. Decompression area is flush with the surrounding scalp and pulsates normally. Reflexes—patellar active, right possibly more than left; no ankle clonus and plantar flexion cannot be elicited upon right foot; abdominal reflexes—right slightly depressed. Fundi—retinal veins slightly enlarged; details of both optic disks clear, though new tissue formation naturally is present. X-ray (Doctor W. H. Stewart)—"bony defect of left frontal area and the oval decompression defect with three silver clips *in situ* are clearly demonstrated; no linear fracture shown" (Fig. 139).

FIGS. 140 AND 141.—Eighteen months following the operation of removal of depressed fracture of left frontal bone and a left subtemporal decompression with marked improvement of the symptoms and signs.

Last Examination (December 2, 1918—18 months after operation).—During the past year, patient has had a general convulsive seizure about every 6 to 8 weeks; in the intervals, however, he is able to hold his position as a watchman and enjoys life in a simple way. The convulsions are not causing a marked mental and emotional deterioration, as they were before the decompression was performed; he no longer has headaches. Decompression area slightly depressed beneath flush of scalp; pulsation normal. Reflexes—patellar active, right possibly more than left; no ankle clonus nor Babinski; abdominal reflexes—right less than left. Fundi—retinal veins slightly enlarged; all details of both optic disks clear and distinct; new tissue formation along optic disk margins and in both physiological cups is still present. Photographs (Figs. 140 and 141) disclose the amount of deformity and disfiguration.

Remarks.—Since the operations in June, 1917, I have examined this patient almost weekly at the hospital clinic and it has been most interesting to observe his mental and emotional improvement; he is now anxious to do some other form of work than that of watchman and his emotional stability has been so benefited that his relatives state he is a "changed man." This patient was operated upon almost as a forlorn hope—merely to give him a possible chance of improvement, and I believe the results obtained up to the present time certainly justify the attempt to improve his condition; from the pathology revealed at the operation, it would be too much to expect an ultimate recovery, but the improvement effected is most encouraging—no matter what the end-result may be.

The above photographs illustrate the small amount of disfiguration of the bony defect in the left frontal area; surely, no silver plate or other foreign body should be inserted for protection.

Although it is most gratifying that the convulsive seizures have lessened both in severity and frequency in this patient (and therefore the operations are justified even though this improvement of the patient's condition is only temporary), it is difficult to conceive that this patient can be so benefited that the convulsions will cease; it would seem that in patients having brain injuries for a long period of years and then finally convulsive seizures do occur, and if these convulsions are permitted to continue for any length of time so that they become at all frequent—more than one a week, that the end-result, operation or no operation, is very doubtful; the lessening of the increased intracranial pressure, however, will in many of these patients delay and retard the rapid progress of the mental and emotional deterioration and from this standpoint alone the operation of subtemporal decompression in selected patients is more than justified.

D. CHRONIC BRAIN INJURIES ASSOCIATED WITH A FRACTURE OF THE BASE OF THE SKULL AND WITH THE SYMPTOMS AND SIGNS PERSISTING; MINOR AND MAJOR EPILEPSY. SUBTEMPORAL DECOMPRESSION.

From what has been stated before, the patients in this series who were operated upon were only selected ones in whom the mental and emotional deterioration had not been severe, the epileptic "habit" not of long duration or severity, and in whom there were marked signs of an increased intracranial pressure which had been ascertained as being primary and not secondary to the convulsions themselves—that is, the increased intracranial pressure was not the result of the convulsions but a possible factor in their causation. These are the patients and the only ones—whether there has been a fracture of the base of the skull or not—for whom the operation of subtemporal decompression can be advised as offering a percentage of these patients a definite chance of relief: naturally, the longer the condition has persisted since the original brain injury, and especially in those conditions complicated by epilepsy in its major form, the less hopeful is the prognosis following any operative procedure.

D. *Chronic brain injuries associated with a fracture of the base of the* *skull; symptoms and signs persisting; minor and major epilepsy. Subtemporal decompression.*

a. *Marked improvement.*

Case 113.—Old severe brain injury associated with a fracture of the skull and with signs of an increased intracranial pressure; convulsive seizures. Right subtemporal decompression. Excellent recovery.

No. 070.—Henry. Twenty years. Colored. Single. Sailor. U. S.

Admitted October 10, 1913—1 year after injury, Polyclinic Hospital. Referred by Doctor J. E. Engelson.

Operation October 21, 1913. Right subtemporal decompression and drainage.

Discharged October 27, 1913—7 days after operation.

Family history negative.

Personal History.—Always well and strong; is not alcoholic. One year ago, patient fell a distance of 20 feet from the main mast down upon the deck, striking upon the top of his head; immediate loss of consciousness and profuse bleeding, with a "watery" discharge, from both ears; upon arrival in port 2 days later, patient was taken to the Marine Hospital, Brooklyn, where he remained unconscious for 8 days; he was discharged on the twelfth day in a stuporous condition and became unconscious upon reaching home. Patient was able to leave the house 2 weeks later, but complained of severe headache; vomited almost daily and 2 months later (9 months ago), patient had his first general convulsive seizure. Beside the severe headaches, dizziness and frequent attacks of vomiting, patient has fainted 2 to 4 times each day during the past 6 months and has had a general convulsive seizure at least once a week; definite blurring of vision.

Examination upon admission (1 year after injury).—Temperature, 98.8°; pulse, 68; respiration, 16; blood-pressure, 148. Well-developed and nourished negro. Complains of severe headache; while sitting upon the bench in the hospital clinic, patient had a convulsive seizure (no localizing signs) and vomited profusely and forcefully—almost projectile in character; pulse descended to 62 during the attack and respirations to 12. Head negative. Hearing negative; otoscopic examination revealed an irregular scar over the lower posterior portion of the right tympanic membrane; left tympanic membrane negative; air conduction greater than bone conduction in both ears. Pupils equal and react normally. Reflexes—patellar increased but equal; no ankle clonus but tendency to a left Babinski; abdominal reflexes not active but equal. Fundi—retinal veins dilated; nasal margins of both optic disks slightly blurred; both optic disks rather pale from new tissue formation in both physiological cups and along the margins—that is, a mild secondary optic atrophy. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 16 mm.); Wassermann test negative and cell count was 5 cells per c.mm. X-ray (Doctor A. J. Quimby)—"no fracture visible."

Treatment.—The definite history of convulsive seizures in a patient having a definite history of fracture of the base of the skull to be followed by spells of *petit mal* and finally by convulsive seizures in a patient having definite signs of an increased intracranial pressure and in the absence of luetic infection—these facts made it advisable to perform a right subtemporal decompression even at this late date in the hope that a lessening of the increased intracranial pressure would diminish the cortical irritation and thereby cause an improvement.

Operation (1 year after injury).—Right subtemporal decompression: usual vertical incision, bone removed, and no complications; removal of bone was rather difficult technically on account of its being rather "ivory" and thick. Dura thickened, fairly vascular and very tense; upon incising it, much cerebrospinal fluid escaped, revealing an edematous cortex which bulged into the dural opening but did not rupture; much arachnoid "sweating." Numerous adhesions between arachnoid and dura and these were severed as far as possible beyond the dural opening. Except for the "wet" condition of the brain (chronic cerebral edema), the cortex was normal. Usual closure with 2 drains of rubber tissue inserted. Duration, 45 minutes.

Post-operative Notes.—Uneventful operative recovery; patient had no spells during hospital residence—headaches disappeared and upon patient's insistence, he was discharged 7 days after operation; incision healing *per primam.*

Examination (May 16, 1914—7 months after operation).—Patient has not had an attack of *petit mal* or a general convulsive seizure since the operation; no headache, and says he feels perfectly well. He began work on board ship 2 weeks after leaving the hospital and has been working ever since; he promises to come for an examination whenever in this port. Reflexes—patellar very active but equal; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi—retinal veins slightly enlarged but not abnormally; details of both optic disks clear; mild secondary optic atrophy is naturally still present.

Examination (October 28, 1915—24 months after operation).—Only one general convulsion since the operation and that occurred following the drinking of gin in Havana 6 months ago; he has had, however, 7 spells of *petit mal*—always following a heavy meal and after severe exertion; last attack was 3 weeks ago. No headache. Decompression area slightly depressed below flush of scalp. Reflexes active but otherwise negative. Fundi—retinal veins within normal limits of size; no edematous blurring of optic disks, which are rather pale, due to the earlier new tissue formation.

Last Examination (March 6, 1918—53 months after operation).—Patient is now in the army transport service and has no complaints at present, although during the past 2 years he has had 6 spells of *petit mal*—not sufficient, however, to make him stop working; no general convulsive seizure during the past 3 years; no headache. Decompression area depressed and pulsates normally. Reflexes active but otherwise negative. Fundi—retinal veins of normal size; all details of both optic disks clear and distinct; mild secondary optic atrophy naturally still present.

Remarks.—It will be necessary to trace this patient over a longer period of time before it can be asserted that the patient is entirely well; the good result obtained so far, however, is undoubtedly due to the comparatively early lessening of the increased intracranial pressure and especially in a person whose intellectual and emotional activities are of a lower order and *skull; symptoms and signs persisting; minor and major epilepsy. Subtemporal decompression.*

a. *Marked improvement.*

Case 113.—Old severe brain injury associated with a fracture of the skull and with signs of an increased intracranial pressure; convulsive seizures. Right subtemporal decompression. Excellent recovery.

No. 070.—Henry. Twenty years. Colored. Single. Sailor. U. S.

Admitted October 10, 1913—1 year after injury, Polyclinic Hospital. Referred by Doctor J. E. Engelson.

Operation October 21, 1913. Right subtemporal decompression and drainage.

Discharged October 27, 1913—7 days after operation.

Family history negative.

Personal History.—Always well and strong; is not alcoholic. One year ago, patient fell a distance of 20 feet from the main mast down upon the deck, striking upon the top of his head; immediate loss of consciousness and profuse bleeding, with a "watery" discharge, from both ears; upon arrival in port 2 days later, patient was taken to the Marine Hospital, Brooklyn, where he remained unconscious for 8 days; he was discharged on the twelfth day in a stuporous condition and became unconscious upon reaching home. Patient was able to leave the house 2 weeks later, but complained of severe headache; vomited almost daily and 2 months later (9 months ago), patient had his first general convulsive seizure. Beside the severe headaches, dizziness and frequent attacks of vomiting, patient has fainted 2 to 4 times each day during the past 6 months and has had a general convulsive seizure at least once a week; definite blurring of vision.

Examination upon admission (1 year after injury).—Temperature, 98.8°; pulse, 68; respiration, 16; blood-pressure, 148. Well-developed and nourished negro. Complains of severe headache; while sitting upon the bench in the hospital clinic, patient had a convulsive seizure (no localizing signs) and vomited profusely and forcefully—almost projectile in character; pulse descended to 62 during the attack and respirations to 12. Head negative. Hearing negative; otoscopic examination revealed an irregular scar over the lower posterior portion of the right tympanic membrane; left tympanic membrane negative; air conduction greater than bone conduction in both ears. Pupils equal and react normally. Reflexes—patellar increased but equal; no ankle clonus but tendency to a left Babinski; abdominal reflexes not active but equal. Fundi—retinal veins dilated; nasal margins of both optic disks slightly blurred; both optic disks rather pale from new tissue formation in both physiological cups and along the margins—that is, a mild secondary optic atrophy. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 16 mm.); Wasserma
tive and cell count was 5 cells per c.mm. X-ray (Doctor A.
"no fracture visible."

Treatment.—The definite history of convulsive seizures
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by spells of *petit mal* and finally by convulsive seizure
definite signs of an increased intracranial pressure

luetic infection—these facts made it advisable to perform a right subtemporal decompression even at this late date in the hope that a lessening of the increased intracranial pressure would diminish the cortical irritation and thereby cause an improvement.

Operation (1 year after injury).—Right subtemporal decompression: usual vertical incision, bone removed, and no complications; removal of bone was rather difficult technically on account of its being rather "ivory" and thick. Dura thickened, fairly vascular and very tense; upon incising it, much cerebrospinal fluid escaped, revealing an edematous cortex which bulged into the dural opening but did not rupture; much arachnoid "sweating." Numerous adhesions between arachnoid and dura and these were severed as far as possible beyond the dural opening. Except for the "wet" condition of the brain (chronic cerebral edema), the cortex was normal. Usual closure with 2 drains of rubber tissue inserted. Duration, 45 minutes.

Post-operative Notes.—Uneventful operative recovery; patient had no spells during hospital residence—headaches disappeared and upon patient's insistence, he was discharged 7 days after operation; incision healing *per primam* possibly more elemental, so that this patient is not subjected to the strain and stress of modern life to which many white patients are subjected; besides, the nervous system of the negro race is not so delicately adjusted as that of the white race and is therefore possibly more stable in withstanding the effects of cortical irritation due to an increased intracranial pressure resulting from traumatic cerebral edema. The bad effect of alcohol upon these patients having an increased irritability of the cerebral cortex has often been observed and its use cannot be too strongly condemned.

The cessation of headache following the operation of decompression in these patients is very impressive; the dural tension, being lessened by the permanent opening and incision of the dura, is undoubtedly the usual cause of the headache.

The complete recovery of hearing following the rupture of the tympanic membrane in these patients occurs in over fifty per cent. of the cases, so that air conduction is greater than bone conduction.

Case 114.—Old severe brain injury associated with a fracture of the base of skull and with signs of an increased intracranial pressure; convulsive seizures and motor aphasia with mild hemiparesis. Left subtemporal decompression. Recovery.

No. 050.—John. Thirty-eight years. White. Married. Laborer. U. S.

Admitted May 16, 1914—7 months after injury, Polyclinic Hospital. Referred by Doctor E. S. Bishop.

Operation May 22, 1914. Left subtemporal decompression and drainage.

Discharged June 4, 1914—12 days after operation.

Family history negative. Patient and all of his relatives are right-handed.

Personal History.—Always well and strong; not alcoholic. Seven months ago, while loading a boat, patient was struck upon the left side of the head by a bucket of coal; unconscious for several minutes and a blood-tinged "watery" fluid trickled from the left ear; was able to walk home but he could not speak for 3 days following the injury; after remaining home for 10 days, patient attempted to work, but on account of the severe continuous headache he was unable to work for more than 2 or 3 days each week. Two months ago, after a series of very severe headaches, he had the first general convulsion; 10 days later, the second general convulsion occurred—beginning in the right side of face, then right arm, right leg and finally the entire body, and it continued for 25 minutes. Since the injury, patient has had a definite impairment of speech—unable to use the proper word at times and even has difficulty in repeating words, although he could write them—the motor type of aphasia. There has been a distinct change of disposition
in that the patient has become more irritable, while the m
definitely impaired, especially for recent events. Duri
convulsions have occurred as frequently as 7 times a d
on the right side of the face and then extending to th
leg and finally a general convulsive seizure occurred
to speak a word for several hours after each atta
of motor aphasia; no agraphia or sensory invo
word deafness, etc.).

Examination upon admission (7 months after injury).—Temperature, 100°; pulse, 80; respiration, 22; blood-pressure, 146. Well-developed and nourished. Unable to speak—merely nods head; considered "stupid" on account of the impairment of speech. Definite weakness of entire right side of body, especially of right side of face and right arm (right facial paresis is of the cortical type in that the right forehead muscles are not involved). Hearing of left ear less acute than that of right; bone conduction equals that of air conduction; otoscopic examination reveals a small scar of a former perforation in the upper posterior portion of the left tympanic membrane. Pupils equal and react normally. Reflexes—patellar exaggerated, right more than left; slight right ankle clonus but no Babinski; abdominal reflexes—right depressed. Fundi—retinal veins dilated; nasal halves of both optic disks blurred by edema—left possibly more than right. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 15 mm.); Wassermann test negative and cell count was 4 cells per c.mm. X-ray (Doctor A. J. Quimby)—"no fracture of the skull is shown."

Treatment.—In the hope that a mechanical lessening of the increased intracranial pressure of this patient would result in an immediate improvement of the condition, so that the headaches would disappear, the convulsions cease, the paraphasia improve and the weakness of the right side of the body become less marked, a left subtemporal decompression was advised.

Operation (May 22, 1914—7 months after injury).—Left subtemporal decompression: usual vertical incision, bone removed, and no complications. Dura thickened and under high tension; upon incising it, a large amount of clear cerebrospinal fluid escaped, revealing an edematous cortex; typical arachnoid "sweating"; the protruding cortex did not rupture and by the end of the operation, the pulsations were normal. Much induration of new tissue formation in the arachnoid and about the cortical veins in the sulci; numerous adhesions between arachnoid and overlying dura and these were severed. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Uneventful operative recovery; within 4 days after operation, patient was able to speak several sentences, using the words in continuity and correctly; convulsive seizure of a mild general type occurred on the seventh day post-operative; no complaint of headache, however, and patient insisted upon being discharged on the twelfth day post-operative; incision healed *per primam.*

Examination (July 6, 1914—44 days after operation).—General improvement continues; no convulsions and no headache except for a "heavy dull feeling" in the head; still paraphasic but much better; slight weakness of right arm, but he no longer limps upon the right leg and no facial weakness can be elicited. Decompression area flush with the surrounding scalp; normal pulsation. Reflexes—patellar active, right greater than left; no ankle clonus and no Babinski; abdominal reflexes—right possibly less active than left. Fundi—retinal veins only slightly enlarged; nasal margins of both optic disks indistinctly blurred.

Examination (October 2

has had only 3 major c

petit mal—momentarily losing consciousness, but of such short duration that patient did not fall. Speech has so improved that he can repeat with only an occasional error the following test phrases: "Around the rugged rock, the ragged rascal ran"; "The third red riding artillery brigade"; "Constantinople"; "Truly rural"; "The sea ceaseth and it rejoiceth us." No agraphia. There is still a slight weakness of the right hand-grip and possibly some awkwardness in performing the various pointing tests, but patient is able to do his work almost as well as before the injury; he tires more easily, however, and requires, he says, 10 hours sleep each night. Reflexes—patellar active, right possibly greater than left; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi negative. Decompression area depressed and pulsates normally.

Last Report (September 16, 1918—52 months after operation).—Patient has had only 2 convulsive seizures during the past 2 years and these, he writes, were only "light ones." He has no marked complaints and works daily. No trouble with speech, although he occasionally skips a word, but "no one notices it." No alcoholism.

Remarks.—An unusually excellent result has up to the present time been obtained in this patient and it has undoubtedly been due to the fact that the cranial injury was a comparatively recent one and that the convulsive seizures were of only 2 months' duration; they were occurring, however, so rapidly during the 3 weeks preceding the operation that the prognosis was very doubtful. This patient was in excellent physical condition and, together with the absence of alcoholism, made the good result possible.

The definite right hemiparesis and motor aphasia with an occasional convulsion beginning in the right arm—these localizing signs were probably due to a cerebral edema as revealed at operation rather than to a supracortical or cortical hemorrhage, which was not disclosed; there could be found no extracranial cause for the development of the cerebral edema and naturally the subarachnoid hemorrhage occurring at the time of the injury must be considered an etiological factor in its production.

A pure motor aphasia is a very rare condition; almost all of these so-called "aphasias" are really variations of paraphasia—there being usually a sensory element in their formation. I do not consider this patient to have had a true condition of motor aphasia.

Case 115.—Old severe brain injury associated with a fracture of the base of the skull and with signs of an increasing intracranial pressure; convulsive seizures. Right subtemporal decompression. Recovery.

No. 173.—Julius. Fifty-two years. White. Married. Tailor. Russia.

Admitted July 2, 1914—8 months after injury, Polyclinic Hospital. Referred by Doctor Alexander Lyle.

Operation July 9, 1914. Right subtemporal decompression and drainage.

Discharged July 26, 1914—17 days after operation.

Family history negative.

Personal History.—Always well and strong; no alcoholism. Eight months ago, patient was knocked down by an automobile; immediate loss of consciousness; profuse bleeding from right ear; taken in an ambulance to a hospital where he remained 2 weeks; unconscious for 2 days. Since his charge from the hospital, patient has had severe constant headaches and a haziness of vision, which has been increasing. Three months ago, patient had the first general convulsion—no localizing signs; second general convulsive seizure occurred 10 days later and during the past 3 months, patient has had 8 convulsions of major character.

Examination upon admission (8 months after injury).—Temperature, 98.2°; pulse, 64; respiration, 30; blood-pressure, 164. Fairly well-developed and nourished. Complains of severe headache and is unable to tell a connected story. Rather "doughy" feel to entire posterior half of scalp with marked tenderness upon pressure. Hearing negative; otoscopic examination reveals a recent scar in the posterior lower quadrant of right tympanic membrane; air conduction greater than bone conduction. No paralyses or impairment of sensation; left-hand grasp possibly weaker than right. Pupils—moderately contracted, equal and react to light sluggishly. Reflexes—patellar very much exaggerated, left greater than right; no ankle clonus but left Babinski; abdominal reflexes—left less active than right (at this point of the examination, the patient suddenly had a convulsion beginning in the left arm, then left leg, and finally the entire body; duration was 2 minutes). Fundi—retinal veins dilated; nasal margins of both optic disks blurred and both nasal halves rather indistinct. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 16 mm.); Wassermann test negative and cell count was 7 cells per c.mm. X-ray (Doctor A. J. Quimby)—"no fracture visible."

Treatment.—The history of cranial injury followed by convulsive seizures and associated with signs of an increased intracranial pressure indicated the advisability of a lowering of the increased intracranial pressure by means of a right subtemporal decompression, in the hope that the cortical irritation could thus be lessened and the convulsive seizures be prevented; to relieve the headache alone would be of great value to the patient. Naturally, if an operation is to be performed now, it would have been much better if the operation had occurred immediately following the injury and thus the great danger of convulsive seizures would have been very much lessened; that is, it is much better judgment to relieve the intracranial pressure early and thus prevent complications rather than to operate upon patients after the complications have occurred—and they do occur in almost 70 per cent. of these non-operated patients having brain injuries associated with the signs of increased intracranial pressure.

Operation (8 months after injury).—Right subtemporal decompression: usual vertical incision, bone removed, and no complications; both the subcutaneous tissue and the temporal muscle were still "boggy" and contained much old blood from the former cranial injury. Dura thickened and under high tension; upon incising it, clear cerebrospinal fluid spurted to a height of 2 cm.; the underlying edematous cortex bulged but did not rupture, as the cerebrospinal fluid escaped in large quantities. Supracortical vessels and arachnoid had a large amount of new tissue formation in and about them—the residue of a former subarachnoid hemorrhage. As the cortex receded and pulsated normally, it was not considered advisable to perform a bilateral decompression, as the pressure had been so greatly lessened by this operation. Usual closure with 2 drains of rubber tissue inserted. Duration, 45 minutes.

Post-operative Notes.—Uneventful operative recovery; several twitches of mild degree occurred in the left arm and left side of face during the operative convalescence, but no general convulsive seizure occurred. Patient felt so well that it was with great difficulty that he could be kept in the hospital until the seventeenth day post-operative when he was discharged; incision healed *per primam.*

Examination (September 12, 1915—14 months after operation).—Patient has had 3 general convulsive seizures since the operation and all occurred during the month of February, 1915—7 months ago; no exciting factor was ascertained as being the cause for these 3 convulsions, but he had complained of headache and dizziness during the preceding week. Patient has been able to work during the past 5 months; no longer complains of headache and has improved both mentally and especially emotionally. No weakness of the arms or legs elicited. Reflexes—patellar active, left greater than right; no ankle clonus but tendency to a left Babinski; abdominal reflexes present and equal. Fundi—retinal veins rather full; only lower nasal margins of both optic disks slightly blurred—other details being distinct. Decompression area slightly depressed below flush of scalp and pulsates normally.

Examination (October 20, 1917—39 months after operation).—During the past 2 years, patient has had 6 general convulsive seizures—2 convulsions occurring on the same day 3 different times; no precipitating cause for these attacks could be ascertained. Patient, however, has been able to work daily and has no complaints except the fear of another attack; no headache nor dizziness. No weakness of the extremities either subjectively or objectively. Decompression area depressed and pulsates normally. Reflexes—patellar active but otherwise negative. Fundi negative, except for slight enlargement of retinal veins.

Last Report (November 20, 1918—52 months after operation).—Son writes that patient has had only 2 convulsive seizures during the past year, but that he has had several "slight faints"—apparently a *petit mal* attack with no muscular contractions. Patient is working daily and has no real complaints except the spells.

Remarks.—Whether this condition of occasional convulsive seizures will gradually subside or whether it will develop into the more serious condition of frequent convulsions cannot be stated with certainty; the latter, however, is to be feared, and especially is this true in patients over 50 years of age in whom arteriosclerotic changes are becoming more and more pronounced.

It would seem that the chronic cerebral edema following the cranial injury and subarachnoid hemorrhage had been sufficient by their irritation to precipitate convulsive seizures, and that the operation of right subtemporal decompression had so lessened this increased intracranial pre re and thereby, to a large extent, the irritative presence of a suprac residue of a former subarachnoid hemorrhage, that the progress o condition had at least been delayed and retarded and it is hoped no cured; from the subsequent history of the patient following the op and especially during the past 2 years, it is doubtful that this latter good result will be obtained.

The rapid recovery of normal hearing of the right ear is very impressive since only 8 months have elapsed since the cranial injury; the fact that the air conduction was greater than the bone conduction within that short period of time indicates that the transmission mechanism of the right middle ear was not permanently damaged.

Case 116.—Old severe brain injury associated with a fracture of the base of the skull and with signs of an increased intracranial pressure; convulsive seizures. Right subtemporal decompression. Improvement.

No. 15.—Richard. Forty-five years. White. Married. Ice-man. Ireland.

Admitted January 5, 1915—10 years after injury, Polyclinic Hospital. Referred by Doctor T. H. Morgan.

Operation January 13, 1915. Right subtemporal decompression and drainage.

Discharged February 5, 1915—23 days after operation.

Family history negative.

Personal History.—Always well and strong; only moderate use of alcohol. Ten years ago, while patient was racing upon a bicycle in Ireland, he fell, striking his head against a large stone; immediate loss of consciousness which persisted for 3 days; blood and a "watery" fluid discharged from the right ear; the patient gradually recovered but severe headaches continued for several months and then subsided. Six years ago (4 years after injury), patient had the first convulsive seizure following an unusually severe headache over a period of one month; no localizing signs. The second spell occurred 6 weeks later, and the third spell one month after the second; they increased both in frequency and in severity until 3 years ago (3 years after the first attack), the patient was having at least one severe seizure every 10 days to 2 weeks; 3 years ago, an osteoplastic flap operation was performed at Bellevue Hospital over the left posterior parietal area; nothing grossly abnormal was noted and the bone-flap was replaced (much difficulty recorded in suturing dura on account of the protrusion of the underlying cortex and the dura could not be closely sutured). Within one month after this operation, the convulsive seizures returned and apparently with greater severity in that the patient would remain unconscious for a period of 3 to 10 hours, to be followed by the most severe headache. Since this time and during the past 3 years, the convulsive seizures have increased in frequency and severity so that he now remains unconscious for a period of 8 to 25 hours following an attack and he has been unable to work during the past year for more than a few days at a time; headaches are extreme and the patient has deteriorated both mentally and emotionally, especially in the past 3 months.

Examination upon admission (10 years after injury).—Temperature, 99°; pulse, 70; respiration, 18; blood-pressure, 142. Large and unusually well-developed man, weighing 204 pounds. Patient is in a rather confused state mentally—this condition being exaggerated as the result of his having had a very severe convulsion 30 hours previously. Many small scars of former scalp lacerations are scattered over his head and also both sides of the tongue are badly lacerated. Over the posterior portion of the left parietal bone is a large horse-shoe incision—the site of the former operation. No paralyses nor impairments of sensation. Hearing negative; air conduction is greater than bone conduction. Pupils equal and react to light normally. Reflexes—patellar exaggerated but equal; no ankle clonus nor Babinski; abdominal reflexes rather depressed but equal. Fundi—retinal veins dilated; nasal halves and temporal margins of both optic disks blurred by edema; physiological cups shallow from new tissue formation. Lumbar puncture—clear cerebrospinal fluid under increased intracranial pressure (approximately 16 mm.); Wassermann test negative and cell count 8 cells per c.mm. X-ray (Doctor A. J. Quimby)—"site of former osteoplastic flap operation over posterior portion of left parietal bone; no line of fracture revealed."

Treatment.—The history of a definite brain injury followed later by convulsive seizures and associated with the signs of a definite increase of the intracranial pressure made advisable the operative relief of this increased intracranial pressure, in the hope that the convulsive seizures could be lessened in frequency and severity and the rapid progress of the condition be at least delayed; naturally, the prognosis was very grave and the operation was performed merely in the belief that if the cortical irritation could be lowered, then a definite improvement would be possible.

Operation (10 years after injury).—Right subtemporal decompression: usual vertical incision, bone removed, and no complications; an unusually difficult operation technically in that the bone was very thick, hard and brittle ("ivory"), and because the temporal muscle was very thick and resistant, making retraction very difficult. Dura whitish and thickened, with many adhesions to the overlying bone; exceedingly tense and upon incising it, clear cerebrospinal fluid welled out, exposing an unusually "wet," edematous cortex which tended to protrude but did not rupture owing to the rapid escape of much cerebrospinal fluid; typical arachnoid "sweating." Along the vessels in the sulci and in the arachnoid itself was a large amount of new tissue formation giving these structures a hazy, cloudy appearance and due most probably to a former subarachnoid hemorrhage. Usual closure with 2 drains of rubber tissue inserted. Duration, 55 minutes.

Post-operative Notes.—Uneventful operative recovery with the exception that upon the seventh day, a small amount of yellowish creamy pus appeared at the lower angle of the incision; a small amount of this pus ("staphylococci") continued to be discharged during the following 7 days when the wound finally healed. (There is no excuse for an infection of this character to occur in a "clean" case; the danger is very great indeed, and it simply means that the operator or the other members of the team have been "dirty," and less frequently so, infective operative material; fortunately in this patient, the infection did not result fatally.) No convulsions occurred during the hospital convalescence and the patient was discharged on the twenty-third day post-operative. Upon arriving home, patient had a slight general convulsive seizure, or rather twitching, lasting only 30 seconds; no mental confusion nor emotional upset, however, followed.

Examination (September 8, 1916—19 months after operation).—Patient has had only 5 general convulsive seizures since the operation and the severity of each attack has become very much lessened; he has never remained unconscious for longer than one hour and has been able to resume his work the same day of the attack; only occasional dull throbbing headache—at which time the decompression area bulges; the patient has been able to work during the past 9 months. The decompression area is slightly depressed and pulsates normally. Reflexes very active but otherwise negative. Fundi—retinal veins enlarged; nasal margins of both optic disks slightly blurred, but the new tissue formation in both physiological cups and along the nasal margins of the optic disks is naturally still present; both disks possibly paler than normal—due probably to new tissue formation.

Last Examination (August 28, 1918—43 months after operation).—Patient has now had 11 general convulsive seizures in all since the operation; no attack, however, during the past 11 months and he feels "in the best of health and spirits." Patient has a heavy dull feeling in the head about every 10 days, but it is most unusual for him to have a headache of any severity—the last one being almost 6 months ago. He has made a marked improvement mentally in that he has become more alert and is able to conduct his ice business much more successfully; no longer loses his temper unless the provocation is extreme. Decompression area is slightly depressed beneath the flush of scalp and pulsates normally. Reflexes active but otherwise negative. Fundi—retinal veins enlarged; nasal margins of both optic disks slightly blurred by edema; new tissue formation naturally persists as at the preceding examination.

Remarks.—The post-operative history of this patient is most instructive and encouraging. The osteoplastic flap operation could naturally be of no permanent benefit to the patient in the presence of the increased intracranial pressure, because, if there was no gross lesion removed at the operation, the dura would have been opened only temporarily and the bone-flap then replaced, and naturally under these conditions there could not have been formed a permanent decompression, so that within a few days after the operation the intracranial pressure would have attained its former degree of pressure and thus the condition would not have been relieved at all to any appreciable degree; besides, the greater risk of this operative procedure, both from a technical standpoint and from the fact that it was performed "high up" over the more highly developed area of the cortex—the motor-sensory region. It is not surprising, therefore, that the convulsions quickly returned and this has been the history of the vast majority of patients having convulsive seizures—whether traumatic or otherwise, who have had cranial operations of the osteoplastic type performed upon them—and especially is this so in the presence of a definite increase of the intracranial pressure. Whether this patient will remain in this improved condition or will gradually return to the former condition of frequent and severe convulsive seizures—only the careful following of his case over a long period of years will tell; if, however, the cortical irritation has been lessened sufficiently by the lowering of the increased intracranial pressure of the cerebral edema by means of the decompression, then the prognosis should be more hopeful and a good result is possible. One, however, cannot allow himself to become too optimistic regarding patients of this character, because they may appear improved for a period of months and even years, and then without any cause that can be ascertained, the patient will undergo a series of convulsive seizures that result most disastrously.

This patient again illustrates the complete recovery of hearing of the right ear even after a discharge of cerebrospinal fluid from it at the time of the injury and thus indicative of a fracture of the base of the skull in this area; in so many patients, this impairment of hearing is only a temporary one—usually not of longer duration than 12 to 18 months.

CASE 117.—Old severe brain injury associated with a fracture of the vault and of the base of the skull and with signs of an increased intracranial pressure; continuous severe headache and spells of *petit mal*. Left subtemporal decompression. Recovery.

No. 292.—Father Francis. Forty-three years. White. Single. Missionary Priest. France.

Admitted June 12, 1915—10 years after injury. Polyclinic Hospital. Referred by Doctor A. B. Duel.

Operation June 22, 1915. Left subtemporal decompression and drainage.

Discharged July 7, 1915—15 days after operation.

Family history negative.

Personal History.—Always well and strong. Ten years ago while working among the Indians in the Canadian Northwest, patient was in a runaway accident in which he was kicked upon the head, left lower jaw fractured and 3 ribs of the left side of his chest broken; patient remained unconscious for one week with no special medical attendance; profuse bleeding from the left ear ceased after 3 days; gradual recovery from the acute condition, and after 5 months patient was able to resume his work in a less active capacity; he always complained, however, of a dull heavy feeling in the head, spells of dizziness and at times a severe headache. Two years ago, during a spell of dizziness, patient "fainted" momentarily and since that time these lapses of consciousness of the *petit mal* type have occurred almost daily; severe frontal and left temporal headaches have increased in frequency and in intensity so that he has been obliged to give up his work during the past 18 months; after any exertion such as walking, a severe pain in the left frontal and left temporal region occurs and will continue until the patient lies down and remains quiet for a period of 30 minutes or more.

Two years ago, in the hope that the left labyrinth might have been involved and was a factor in causing the condition, a left radical mastoid operation was performed in Hartford but no marked improvement resulted. Five months ago, two trephine openings over the left front-parietal area were made in a hospital in Springfield in the "belief" that they would, in some way not to be explained, improve the condition; merely 2 buttons of bone removed, the dura not opened and naturally no benefit was obtained (it is possible that an effort was being made to locate a fracture of the skull which, as is now well known, is possibly the most unimportant part in the diagnosis and especially in the treatment of brain injuries).

Examination upon admission (10 years after injury).—Temperature, 98.8°; pulse, 68; respiration, 16; blood-pressure, 142. Well-nourished and developed; rather anxious facies; depressed and melancholic, fearing that he will never be able to return to his work. Mentality excellent. Examination of head is negative, except for scars of former left mastoid operation and the two trephine openings over the left posterior frontal and left parietal areas, respectively; no pulsation palpable. Hearing of the left ear is markedly impaired; bone conduction is greater than air conduction. Pupils equal and react normally. Reflexes—patellar very much exaggerated, right possibly more active than left; exhaustible right ankle clonus and suggestive right Babinski; abdominal reflexes—right less active than left. Fundi—retinal veins rather full; nasal margins and a small portion of both nasal halves of optic disks obscured by edema; both physiological cups shallow from new tissue formation and a small amount of new tissue about the temporal margins of optic disks. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 15 mm.); Wassermann test negative and cell count was 4 cells per c.mm. X-ray (Doctor A. J. Quimby)—"left mastoid area blurred with new bone formation; irregular bony defects with new bone formation over the left frontal area posteriorly. Indistinct and irregular line of fracture extending transversely through the squamous portion of the left temporal bone."

Treatment.—A left subtemporal decompression was considered advisable to lessen the increased intracranial pressure and thereby relieve the headache and thus possibly lessen the cortical irritability so that the spells of *petit mal* would cease, the vertigo disappear and the patient be benefited.

Operation (10 years after injury).—Left subtemporal decompression: rather high vertical incision, bone removed, and no complications; much fibrous tissue in the fibres of the temporal muscle, due probably to a former hemorrhage in the muscle itself beneath the temporal fascia; this was confirmed largely by finding an irregular transverse fracture ridge extending through the lower portion of the underlying squamous bone, in which situation the underlying left middle meningeal artery could have been easily torn at the time of the fracture. Dura was thickened and under high tension; upon incising it, much cerebrospinal fluid escaped, revealing a very edematous swollen cortex which tended to protrude but did not rupture. Slight hazy induration about the vessels in the sulci and in the arachnoid itself—the residue of a former subarachnoid hemorrhage. No gross lesions in or upon the cortex visible. At the end of the operation, the cortex had receded and pulsated almost normally. No adhesions between arachnoid and the overlying dura observed. Usual closure with 2 drains of rubber tissue inserted. Duration, 50 minutes.

Post-operative Notes.—Uneventful operative recovery; within 4 days after operation, the dull heavy feeling in the head had lessened and patient said that "my head feels better than it has for months." Patient was discharged on the fifteenth day after operation; incision healed *per primam.*

Examination (April 7, 1917—22 months after operation).—Patient has had only 6 spells of *petit mal* since the operation, although he has had almost each month an acute attack of dizziness and "heaviness" in the head but "nothing happens"; no longer has severe headache following mild exertion and is able to perform his duties at La Salette College, Hartford; no longer is depressed and melancholic. Decompression area is slightly depressed and pulsates normally. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—retinal veins possibly slightly enlarged; slight edematous blurring of lower quadrant of nasal margins of both optic disks; new tissue formation naturally persists in both physiological cups and along the margins of the optic disks. Second X-ray (Doctor A. J. Quimby)—"in addition to the first plate, the circular decompression opening is shown" (Fig. 142).

Last Examination (February 20, 1919—55 months after operation).—Patient has not had a "fainting spell" for 14 months and has only had 13 in all since the operation; occasional headache of mild severity, but he is otherwise well and able to perform his duties daily; no dizziness. Patient says: "Place where you operated is now always sunken in and I can feel it beating." Reflexes active but otherwise negative. Fundi negative, except for the new tissue formation.

Remarks.—It would seem that a most satisfactory result had been obtained in this patient and that the operation to improve his condition was fully justified. I am of the opinion, however, that he cannot be considered cured until a longer period of time has elapsed, although apparently he has now entirely recovered from the condition. It is hard to conceive that a patient can have even a small amount of fibrous residue upon the cortex resulting from a former subarachnoid hemorrhage and yet, even in the absence of a definite increase of the intracranial pressure, that the patient can be normal both mentally and physically, and also emotionally; this latter complication of emotional instability is most easily induced and the return to normal is most difficult.

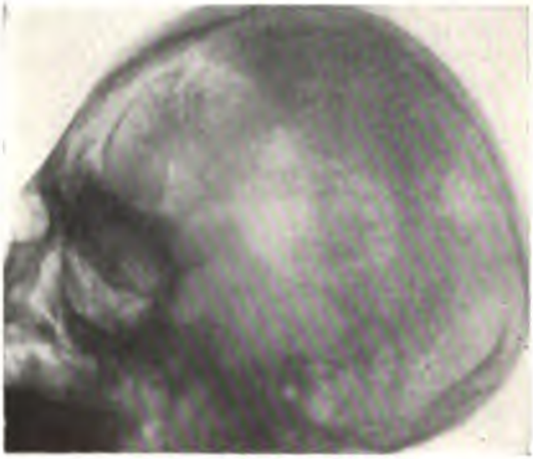

FIG. 142.—New bone formation following an old depressed fracture of the left frontal area in a patient with persisting symptoms and signs, which disappeared following a left subtemporal decompression.

The former trephine openings and merely small "buttons" of bone removed and the dura not opened may justly be termed meddlesome surgery; the purpose of such inadequate and even not exploratory operations are, to be sure, of very little risk to the patient (the dura not being opened), but also they are of no possible value to the patient; they may be "operations" and it may be added, "useless operations." The r grams disclosing the bony defects and new bone formation in frontal area are interesting; it would seem that a larger area of b been removed at the earlier operations.

CASE 118.—Old severe brain injury associated with a fracture base of the skull and with signs of an increased intracranial p convulsive seizures. Right subtemporal decompression. Improvement.

No. 427.—John. Twenty-three years. White. Married. Mechanic. U. S.

Admitted November 11, 1915—8 years after injury. Polyclinic Hospital. Referred by Doctor E. C. Douglas.

Operation November 22, 1915. Right subtemporal decompression and drainage.

Discharged December 19, 1915—27 days after operation.

Family history negative.

Personal History.—Always well and strong. Eight years ago while playing baseball, patient was struck upon the right side of the head by a batted ball; no complete loss of consciousness—merely stunned, but owing to the severe headache he was unable to continue playing; one hour later he walked home and lay down upon a couch; a small amount of watery fluid discharged from the right ear. Twelve hours later, a general convulsive seizure occurred, the patient remaining unconscious for 12 hours; with the exception of rather severe headaches, he made apparently a normal and uneventful recovery so that he was able to return to school after 10 days; almost daily headaches, however, of mild severity continued, but the patient was considered well until 6 months later, when he had a second general convulsive seizure, and the third attack occurred 5 months later: at this time, the condition was diagnosed as one of epilepsy and his treatment consisted of a small amount of bromide after each meal. The fourth convulsion occurred 6 months later, and since that time and during the past 6 years, the patient has had a general convulsive seizure every 3 to 6 months, until during the past year the attacks have increased in frequency to one every month of a severe character and *petit mal* attacks on the average of one every 2 weeks; during the severe major attacks, patient has several times dislocated his right arm at the shoulder, but has required a doctor to reduce the dislocation only twice—being able to reduce it himself the other times. Beside the convulsive seizures, patient now complains of a dull heavy frontal headache, loss of ambition—"always tired," and unable to remember things, especially recent occurrences; he has been unable to work during the past 8 months—"nobody will take me."

Examination upon admission (8 years after injury).—Temperature, 98.6°; pulse, 76; respiration, 18; blood-pressure, 128. Well-nourished and developed. Mentality rather retarded and confused; depressed and melancholic—"no hope for me." No external evidence of former head injury. Hearing negative; air conduction greater than bone conduction in both ears; otoscopic examination negative. Pupils equal and react normally. Reflexes—patellar very much exaggerated but equal; no ankle clonus nor Babinski; abdominal reflexes both depressed but equal. Fundi (Doctor J. A. Kearney)—"vision normal, media clear, disks circular; temporal margins of disks distinct, while nasal halves are blurred by edema; retinal veins engorged. Irregular heaping of pigment interspersed with lighter streaks over entire fundus; both eyes about the same." Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 16 mm.); Wassermann test negative and cell count was 7 cells per c.mm. X-ray (Doctor A. J. Quimby)—"no sign of fracture visible."

Treatment.—The definite history of cranial injury sufficient to cause a discharge of cerebrospinal fluid from the right ear and therefore associated with a fracture of the base of the skull, and followed within 12 hours by a convulsive seizure which has recurred at varying intervals during the past 8 years with increasing frequency and severity, and now the demonstration of an increased intracranial pressure by the ophthalmoscope and at lumbar puncture—these data make the operation of cranial decompression advisable and especially so since the patient has had competent medical treatment—at least during the past 6 years.

Operation (8 years after injury).—Right subtemporal decompression: usual incision, bone removed, and no complication. Dura very much thickened and under moderate tension; upon incising it, clear cerebrospinal fluid welled out, and upon enlarging the dural opening the underlying cortex bulged markedly; over the entire cortex exposed was a cloudy grayish cystic formation, at least one-fourth inch in thickness—most probably resulting from the organization of a former subdural hemorrhage; upon "knicking" this cystic formation, a pale straw-colored fluid escaped, permitting the cyst to collapse; an area of over one inch of its outer wall was excised. About the supracortical vessels in the sulci and in the arachnoid itself was a cloudy induration of new tissue formation—the residue of a former subarachnoid hemorrhage. At the end of the operation, the cortex had receded sufficiently to pulsate almost normally, so that the closure of the overlying temporal muscle was facilitated. Usual closure with 2 drains of rubber tissue inserted. Duration, one hour.

Post-operative Notes.—Convalescence very stormy in that a post-operative pneumonia of the right lung developed on the third day and in addition the diagnosis of either an hepatic or subdiaphragmatic abscess was made, but the latter diagnosis could not be confirmed; patient under care of Doctors Alexander Lyle and Ernest Bishop and after an eventful period of 2 weeks, he made an excellent recovery so that he was discharged on the twenty-seventh day after operation. No convulsions occurred during the hospital residence, although patient complained of a dull frontal headache throughout this period; at discharge, the decompression area bulged rather tensely beyond the flush of scalp but it pulsates normally.

Examination (April 10, 1917—17 months after operation).—Patient has had in all only 7 major convulsive seizures and 10 minor spells with only momentary loss of consciousness; he has improved markedly both mentally and emotionally, works daily, and with the exception of the occasional seizure of dull headache, patient considers himself "in fine condition." Reflexes active but otherwise negative. Fundi—retinal veins slightly enlarged; both fundi rather edematous but not localized to the optic disks, which are clear and distinct. Decompression area slightly depressed the flush of scalp and pulsates normally.

Last Examination (February 5, 1919—39 months after oper
A major convulsive seizure now occurs on the average of once every
and a *petit mal* attack about once a month: the effect of these sp
the patient does not appear to be very harmful (as before the opera
that the patient is only "knocked out," as he says, for a period of

hours—he then being able to resume his work and with no real discomfort; the headache is never severe—"they don't bother me at all." Decompression area slightly depressed beneath flush of scalp and the pulsation is normal. Reflexes active but otherwise negative. Fundi—retinal veins still slightly enlarged; both retinæ suffused and congested, but no edematous blurring of details of optic disks themselves.

Remarks.—In this patient, the convulsive condition has undoubtedly been improved and at least delayed, but at any moment and within the near future this patient may develop a series of convulsive seizures and rapidly deteriorate. In the majority of patients, and especially of this type where there still persists undoubtedly a chronic cerebral edema, both resulting from the convulsions themselves and due also to the irritative presence of the supracortical residue of the former hemorrhage—in these patients the danger of an increased frequency and severity of the convulsions is very great indeed; it would seem that in some of these patients there is present a mild external hydrocephalus (and thus the cause of the chronic cerebral edema), due to a partial blockage of the excretion of the cerebrospinal fluid through the normal stomata of exit in the cortical veins, sinuses, etc., which have become blocked to a greater or less extent by the new tissue formation resulting from the organization of the supracortical hemorrhage. It might be advisable to perform a bilateral decompression upon patients of this character when or in whom a unilateral decompression does not seem to be sufficient both in relieving the increased intracranial pressure and as a means of increased drainage of the blocked cerebrospinal fluid.

Case 119.—Old severe brain injury associated with a fracture of the skull and with signs of an increased intracranial pressure; convulsive seizures. Left subtemporal decompression. Improvement.

No. 447.—John. Thirty-one years. White. Married. Policeman. U. S.

Admitted November 16, 1915—3½ years after injury. Polyclinic Hospital. Referred by Doctor J. W. Brannan.

Operation December 1, 1915. Left subtemporal decompression and drainage.

Discharged December 21, 1915—20 days after operation.

Family history negative.

Personal History.—Always well and strong; of good habits. Three and a half years ago (June 12, 1912), while riding to a fire as a mounted policeman, patient was thrown from his horse; immediate loss of consciousness; profuse bleeding from right ear; taken to the Flushing Hospital in an ambulance and a depressed fracture of right frontal bone removed (dura, however, not being opened); patient remained unconscious for 19 days; gradual recovery occurred so that he was finally discharged from the hospital in September, 1912 (3 months after injury), and appeared to be in good condition. Patient was able to return to his work on "light duty" and with the exception of almost daily headaches of moderate severity, he considered himself well. Twelve months after injury (2½ years ago), the first general convulsive seizure occurred with loss of consciousness, biting of tongue and sphincteric relaxation; headaches increased both in frequency and severity after the convulsive seizure and he began to "lose interest in things." The second convulsion occurred 2 months later, and during the past 2 years a major convulsion has occurred on an average of every 6 weeks or 2 months; definite mental and emotional impairment, so that the patient has been unable to work during the past 6 months; he has lost all confidence in himself and becomes so irritable that his relatives find it very difficult to live in the same house with him. There is an indefinite history that the convulsions always begin on the right side of the body, either in the right arm or right leg, but no competent observer confirms this.

Examination upon admission (3½ years after the injury).—Temperature, 98.8° ; pulse, 72 ; respiration, 16 ; blood-pressure, 144. Well-developed and nourished; very much depressed and says "I want to end it all." Over the right frontal area is an irregular scar and a slight depression of a bone defect; no pulsation palpable. Hearing negative; air conduction greater than bone conduction in both ears; indefinite and irregular thickening of posterior portion of right tympanic membrane—scar tissue of a former laceration in this area at the time of the head injury. Pupils equal and react normally. Reflexes—patellar active but equal; no ankle clonus but suggestive right Babinski; abdominal reflexes—right possibly less active than left. Fundi—retinal veins enlarged; nasal margins of both optic disks blurred, but other details of optic disks clear and distinct. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 14 mm.) ; Wassermann test negative and cell count was 4 cells per c.mm. X-ray (Doctor A. J. Quimby)—"irregular bony defect of 3 cm. in diameter in right frontal area; new tissue formation about the periphery and over the underlying dura. No line of fracture can be seen."

Treatment.—The definite history of cranial injury followed within a year by convulsive seizures and associated, as now demonstrated, by an increased intracranial pressure and the neurological examination revealing signs indicative of a lesion affecting the left hemisphere possibly more than the right—these data made a left subtemporal decompression advisable in the hope that the convulsive seizures might thus be lessened, the headache relieved and the general condition of the patient be improved.

Operation (3½ years after injury).—Left subtemporal decompression: usual vertical incision, bone removed, and no complications. Dura thickened, vascular and very tense; upon incising it, clear cerebrospinal fluid spurted to a height of one inch; upon enlarging the dural opening, there were exposed and severed many adhesions between the cortex and the overlying dura and in the upper portion of the left temporal lobe was an old laceration of the cortex extending backward beyond the posterior edge of the bony decompression opening; a bluish cystic mass lay within the cortical laceration and over the lower half of the left temporal lobe was a bluish cystic formation—the size of a silver quarter; both these hemorrhagic cysts were incised, allowing a straw-colored fluid to escape. Many adhesions were severed as widely as possible about the decompression area. At the end of the operation, the cortex had receded and pulsated almost normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 45 minutes.

Post-operative Notes.—Uneventful operative recovery; no convulsions occurred during hospital residence and at discharge on the twentieth day after operation, the headache was less than at any time during the preceding 6 months; incision healed *per primam.*

Examination (January 10, 1917—13 months after operation).—Patient has had six general convulsive seizures since leaving the hospital—five of these occurring within the first seven months. Patient has been on "light duty" during the past 6 months, no longer complains of severe headaches and his general mental and emotional condition has so improved that his wife says "he is like his ownself again." Decompression area is definitely depressed and the pulsation is normal. Reflexes active but otherwise negative. Fundi—retinal veins of normal size; both optic disks clear and distinct but surrounding retinæ are slightly suffused and congested with a "pepper-pot" appearance (report of Doctor J. A. Kearney). A second X-ray by Doctor A. J. Quimby—"similar appearance as in former röntgenogram with addition of the left decompression bony defect" (Fig. 143).

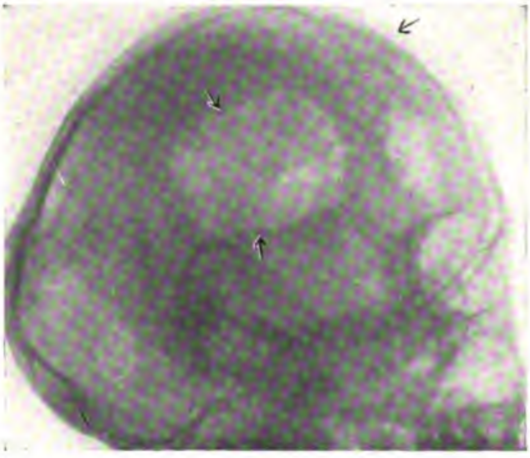

Fig. 143.—Large irregular bony defect of right frontal area, following a depressed fracture and associated with convulsive seizures. Marked improvement following the lowering of the increased intracranial pressure by means of a left subtemporal decompression.

Last Report (November 16, 1918—35 months after operation).—During the past 2 years, patient has had a general convulsive seizure every 4 months; these attacks are not as severe as formerly and their effect does not last longer than 2 hours. Patient is still on "light duty" and as the spells usually occur at night, his work is thereby not affected. No complaints of severe headache, and if it were not for the convulsive seizures he would consider himself a "well man."

Remarks.—The pathology of cortical laceration as revealed at the left subtemporal decompression would make it appear that this lesion was one of *contre-coup* in that the area of contact was in the right frontal region at the site of the depressed bone. These *contre-coup* brain injuries are of common occurrence, and it is of importance in each case to examine the patient neurologically most carefully in the hope that if there is present an increased intracranial pressure making a subtemporal decompression advisable, then there will be ascertained signs pointing to the hemisphere more involved. The chief function of the operation, however, is naturally to lessen the increased intracranial pressure and if possible upon the side more affected; if there are no localizing signs to be elicited, then the right subtemporal decompression is to be preferred in right-handed patients and thus the slight danger of any possible damage to the neighboring motor speech area in the left cerebral cortex be avoided; usually in right-handed patients, and particularly if the parents and grandparents were also right-handed, then the motor speech area is usually in the posterior portion of the third left frontal convolution (Broca's motor speech center).

This patient has been undoubtedly improved as the result of the operation, but whether the improvement will continue over a longer period of years cannot be assured; he naturally should lead a rather careful hygienic life with no prolonged mental and emotional strains, and a restricted light diet with no meat or meat soup, tea, coffee, and particularly alcohol—that is, a vegetarian diet with the addition of not more than 2 eggs a day, 2 glasses of milk and the white of chicken not more than twice a week; fish is permissible.

CASE 120.—Old severe brain injury associated with a fracture of the occipital bone and with signs of an increased intracranial pressure; convulsive seizures. Right subtemporal decompression. Improvement.

No. 1004.—James. Twenty-three years. White. Single. Mechanic. U. S.

Admitted August 1, 1918—10 years after injury. Polyclinic Hospital. Referred by Doctor Charles Coburn, Philadelphia.

Operation August 12, 1918. Right subtemporal decompression and drainage.

Discharged August 31, 1918—19 days after operation.

Family history negative.

Personal History.—Always well and strong. Ten years ago, patient was struck over the head by a baseball bat; only momentary loss of consciousness and a trickling of clear watery fluid from the right ear; he was able to walk home, and after 4 days there were no complaints. Patient was considered a normal child until 5 years ago (that is, 5 years after the head injury), when the first general convulsive seizure occurred; no localizing signs and no definite persistent complaint of headache or other symptoms; the second general convulsion occurred 6 months later, and the third convulsive seizure followed in 4 months. During the past 4 years, these convulsive seizures of general character have increased both in severity and frequency, so that they are occurring now every 4 to 6 weeks—the last spell being 2 days ago. During the past 6 months, patient had an indefinite headache of moderate severity; he has become emotionally unstable, sluggish mentally and unable to work during the past 3 months. The patient has had competent medical treatment.

Examination upon admission (10 years after injury).—Temperature, 98.6°; pulse, 78; respiration, 20; blood-pressure, 132. Fairly well-developed and nourished; general mental and emotional condition unusually good for a patient who has had convulsive seizures during a period of 5 years. No external evidence of cranial injury. Hearing less acute in right ear—bone conduction being almost equal to air conduction in the right ear; otoscopic examination negative. Pupils equal and react normally. Reflexes very active but otherwise negative. Fundi—retinal veins slightly enlarged; indistinct edematous blurring of the lower nasal margins of both optic disks. Lumbar puncture—clear cerebrospinal fluid under increased pressure (14 mm.); Wassermann test negative and cell count was 4 cells per c.mm. X-ray

(Doctor G. W. Welton)—"two distinct and irregular lines of fracture in the right occipital bone" (Fig. 144).

Treatment.—The definite history of cranial injury sufficient to fracture the skull so that cerebrospinal fluid escaped from the right ear and then followed, after an interval of 5 years, by general convulsive seizures of increasing severity and frequency, the condition not being improved by competent medical treatment, and now the signs of an increased intracranial pressure being present and the former fracture of the skull confirmed by röntgenograms—the only chance apparently that this patient has of being improved or the condition being delayed or retarded is by means of a subtemporal decompression to lessen mechanically the increased intracranial pressure and thus diminish the cortical irritability—at least temporarily. The patient being right-handed as were also his parents and grandparents, and there being no localizing signs ascertainable by careful neurological examinations, the subtemporal decompression was performed on the right side of the skull.

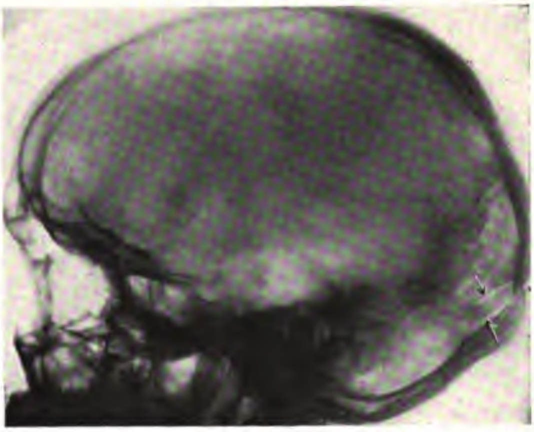

FIG. 144.—Two irregular linear fractures of right occipital bone in a patient following an old cranial injury with convulsive seizures. Definite improvement following a right subtemporal decompression.

Operation (10 years after injury).—Right subtemporal decompression: usual vertical incision, bone removed, and no complications. Dura very thick, whitish and tense; upon incising it, clear cerebrospinal fluid spurted to a height of 2 cm. and upon enlarging the dural opening, the underlying cerebral cortex bulged but did not rupture owing to the rapid escape of much cerebrospinal fluid. Cortex very much congested and very edematous with much new-formed tissue in sulci and in the overlying arachnoid—the residue of a former subarachnoid hemorrhage. No gross cortical laceration or hemorrhagic cyst visible. At the end of the operation, the cortex had receded and pulsated normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 35 minutes.

Post-operative Notes.—Uneventful operative recovery; no convulsions during the hospital residence and the patient was discharged on the nineteenth day after operation; incision healed *per primam*.

Last Report (January 12, 1919—5 months after operation—letter from mother).—Patient had one general convulsive seizure of light character 2 months after operation; after one hour, he was able to continue his work and did not complain of the usual headache, nausea or vomiting. Since then, he has had no complaints and works daily.

Remarks.—This recent case is reported to emphasize the necessity of most careful neurological examinations in order to ascertain definitely preconvulsive stage may have the motor convulsive seizures precipitated by a cranial injury of greater or less severity and naturally in these patients a differential diagnosis is very difficult and, undoubtedly at times, impossible, especially if the signs due to the cranial injury complicate the picture of an idiopathic type of epilepsy.

This case is of too recent date to permit an opinion regarding the ultimate prognosis, but it is encouraging that 5 months could have elapsed with only one convulsion occurring. A further report of this patient will be made later in detail.

b. *No marked improvement.*

Case 121.—Old severe brain injury associated with a fracture of base of skull and with signs of an increased intracranial pressure; severe headache and convulsive seizures. Left subtemporal decompression. Improvement of headache but only a temporary lessening of the convulsions.

No. 245.—Abe. Fifteen years. White. Student. U. S.

Admitted January 31, 1914—6 years after injury. Polyclinic Hospital. Referred by Doctor A. F. Stoloff.

Operation February 5, 1914. Left subtemporal decompression and drainage.

Discharged February 14, 1914—8 days after operation.

Family history negative.

Personal History.—Always well and strong. Six years ago, patient was struck over the head by a large glass decanter; only momentary loss of consciousness and was able to walk home; complained of severe headache and dizziness; blood-tinged watery fluid discharged from the left external auditory canal for a period of only 24 hours; patient was able to go to school the following day and after one week, he no longer complained of headache. Two months later, the first general convulsive seizure occurred; within 2 years, however, patient was having as many as 3 or 4 convulsions each night—almost all of them being nocturnal. During the past 2 years, the convulsive seizures frequently began in the right hand or in the right side of the face and then became general in character; he has had as many as 8 within 24 hours. During the past 6 months, patient has complained of persistent frontal headache and within the last 3 months, there has developed a definite paraphasia. Competent medical treatment has been of no benefit to the patient.

Examination upon admission (8 years after injury).—Temperature, 98.8°; pulse, 74; respiration, 18; blood-pressure, 126. Well-developed and nourished. Except for a scar over the left posterior frontal area, there are no other signs of former cranial injury. Hearing negative; otoscopic examination negative. Definite weakness of right arm, but no weakness of the right side of face or of the right leg. Some paraphasia in that the patient cannot repeat the test phrases correctly. Pupils equal and react normally. Reflexes—patellar very active, right greater than left; no ankle clonus but tendency to right Babinski; abdominal reflexes—right depressed. Fundi—retinal vessels dilated; nasal margins of both optic disks obscured by edema; no new tissue formation observed. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 15 mm.); Wassermann test whether the convulsive seizures are of the so-called idiopathic type (and they usually are), in which no definite organic lesion can be considered an etiological factor and for this type of epilepsy little if anything can be offered, or if there is a definite organic lesion, intracranial or otherwise, which is the primary cause of the condition. In a small percentage of these patients having convulsive seizures, there are, just as in this patient, signs of an increased intracranial pressure and these patients alone are the only ones upon whom a cranial decompression can be considered, and especially if the original cause of the condition was a brain injury, tumor, etc. The increased pressure, however, may be secondary to frequent convulsive seizures which have caused a mild cerebral edema, but this can be decided in most patients by preventing the convulsions from occurring for a period of 4 to 6 weeks by the vigorous use of bromides, etc., and then if the ophthalmoscope and the spinal mercurial manometer still demonstrate the presence of a definite increase of the intracranial pressure, then this pressure must be considered as a primary factor rather than a secondary one to the convulsive seizures.

This patient was at no time carefully examined neurologically during the 5 years preceding the operation—he was considered merely "an epileptic" and no credence given to the possibility that the former cranial injury might have been a factor in producing the condition; no careful ophthalmoscopic examinations were at any time made and by no means a lumbar puncture and especially the registration of the pressure of the cerebrospinal fluid. (There is still a belief among many physicians that a lumbar puncture is a dangerous procedure and should only be resorted to in the presence of an acute severe illness where the immediate danger is great—as in meningitis and similar conditions. A lumbar puncture, properly performed with the usual asepsis and regard for removing only a small amount of the cerebrospinal fluid for cytological examinations and the Wassermann test, is not a dangerous procedure and should always be performed for any intracranial condition or disease, or supposed disease of the cerebrospinal system.) If an X-ray had been taken, naturally more attention would have been given to the history of the former injury, but merely because most of us have had a cranial injury of greater or less severity in our youth and no convulsions result and also almost all patients having the so-called idiopathic epilepsy have similar histories of cranial injuries which are not the cause of the convulsive seizures, it is very natural for the medical profession to become very sceptical regarding cranial traumata as a factor in the production of convulsive seizures, but these patients having definite cranial injuries should at least be given the benefit of careful neurological examinations and particularly in respect to the presence or not of an increased intracranial pressure by careful ophthalmoscopic examinations and,
accurate of all, the measurement of the pressure of the cerebrosp
fluid at lumbar puncture by means of the spinal mercurial manometer.
patient having convulsive seizures should be considered carefully
competently examined, unless a lumbar puncture
both the pressure and cytological character of
been made. It is granted that patients havi

preconvulsive stage may have the motor convulsive seizures precipitated by a cranial injury of greater or less severity and naturally in these patients a differential diagnosis is very difficult and, undoubtedly at times, impossible, especially if the signs due to the cranial injury complicate the picture of an idiopathic type of epilepsy.

This case is of too recent date to permit an opinion regarding the ultimate prognosis, but it is encouraging that 5 months could have elapsed with only one convulsion occurring. A further report of this patient will be made later in detail.

b. *No marked improvement.*

Case 121.—Old severe brain injury associated with a fracture of base of skull and with signs of an increased intracranial pressure; severe headache and convulsive seizures. Left subtemporal decompression. Improvement of headache but only a temporary lessening of the convulsions.

No. 245.—Abe. Fifteen years. White. Student. U. S.

Admitted January 31, 1914—6 years after injury. Polyclinic Hospital. Referred by Doctor A. F. Stoloff.

Operation February 5, 1914. Left subtemporal decompression and drainage.

Discharged February 14, 1914—8 days after operation.

Family history negative.

Personal History.—Always well and strong. Six years ago, patient was struck over the head by a large glass decanter; only momentary loss of consciousness and was able to walk home; complained of severe headache and dizziness; blood-tinged watery fluid discharged from the left external auditory canal for a period of only 24 hours; patient was able to go to school the following day and after one week, he no longer complained of headache. Two months later, the first general convulsive seizure occurred; within 2 years, however, patient was having as many as 3 or 4 convulsions each night—almost all of them being nocturnal. During the past 2 years, the convulsive seizures frequently began in the right hand or in the right side of the face and then became general in character; he has had as many as 8 within 24 hours. During the past 6 months, patient has complained of persistent frontal headache and within the last 3 months, there has developed a definite paraphasia. Competent medical treatment has been of no benefit to the patient.

Examination upon admission (8 years after injury).—Temperature, 98.8°; pulse, 74; respiration, 18; blood-pressure, 126. Well-developed and nourished. Except for a scar over the left posterior frontal area, there are no other signs of former cranial injury. Hearing negative; otoscopic examination negative. Definite weakness of right arm, but no weakness of the right side of face or of the right leg. Some paraphasia in that the patient cannot repeat the test phrases correctly. Pupils equal and react normally. Reflexes—patellar very active, right greater than left; no ankle clonus but tendency to right Babinski; abdominal reflexes—right depressed. Fundi—retinal vessels dilated; nasal margins of both optic disks obscured by edema; no new tissue formation observed. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 15 mm.); Wassermann test negative and cell count was 4 cells per c.mm. X-ray (Doctor A. J. Quimby) —"no fracture of the skull observed."

Treatment.—A left subtemporal decompression was considered advisable in the hope that a lessening of the increased intracranial pressure would permit an improvement not only of the headache but also of the convulsive seizures; although the chances were against the patient being permanently benefited, yet the operation was considered justified even at this late date, and naturally, since the patient had had so many convulsive seizures, the prognosis was most grave.

Operation (6 years after injury).—Left subtemporal decompression: usual vertical incision (slightly anterior), removal of bone, and no complications; bone itself was unusually thick and vascular. Dura thickened, fibrous and very tense; upon incising it, the underlying edematous cortex tended to protrude; upon enlarging the dural opening, bluish white areas of organized blood-clot were exposed in the sulci about the vessels, being apparently fibrous and cystic formations—the residue of a former subarachnoid hemorrhage; numerous adhesions between the arachnoid of cortex and the overlying dura, and these were severed as widely as possible. The escape of cerebrospinal fluid permitted the cortex to recede and to pulsate almost normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 45 minutes.

Post-operative Notes.—Uneventful operative recovery; except for a "fulness" in the head, the patient had no complaints; he was discharged on the eighth day post-operative—no convulsions having occurred; incision healed *per primam.*

Examination (March 20, 1915—13 months after operation).—Patient did not have a convulsive seizure until 3 months ago (10 months after operation), when the first spell occurred while he was performing upon the stage; since then, 2 other convulsive seizures have occurred at night in bed. Patient feels well, as he no longer has severe headaches nor dizziness; also no trouble with speech. No distinct weakness of the right arm can be ascertained. Decompression area is flush with the surrounding scalp and pulsates normally. Reflexes—patellar very active, right possibly being slightly greater than left; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi—retinal vessels slightly enlarged; lower nasal margins of both optic disks only slightly blurred by edema.

Examination (October 16, 1917—44 months after operation).—Patient has had a stormy period during the past 2 years; although he has not complained of headache and has been able to work upon the stage regularly, yet a convulsive seizure occurs on an average of once every 3 weeks; the after-effects, however, are of only short duration and the patient's mental and emotional make-up has apparently not been damaged. Decompression area is only slightly depressed beneath flush of scalp and patient says it bulges at times; normal pulsation. Reflexes—patellar very active, right still possibly a little more active than left; otherwise negative. Fundi—retinal veins slightly enlarged; the indefinite edematous blurring of the lower nasal margins of both optic disks is still present.

Last Report (December 2, 1918—58 months after operation).—D the past year, patient has had 2 severe attacks of acute nephritis and during each of these periods, the convulsive seizures have become much more severe and frequent. He no longer suffers from headache and is able to perform upon the stage. "If it were not for the convulsions, I would be a well man. The wound never really sinks in."

Remarks.—The ultimate prognosis for this patient is naturally bad. The operation undoubtedly delayed and retarded the progress of the convulsive seizures and the headache has been relieved, but a rapid return of the frequency and severity of the convulsions may be expected, and especially when complicated by the condition of nephritis. As the increased intracranial pressure has not been entirely relieved by the operation of decompression (unilateral), in that the operative area never became really depressed as it should have become within 6 months after the operation and to a depth of usually 1 cm., it might have been advisable to have performed a bilateral decompression within a year following the first operation and this greater relief of the increased intracranial pressure might have obtained a better result; if a similar patient should present himself now, I should advise this method of procedure. Taken in all, however, this patient was hardly a fair test for the operation in that his condition was a rather extreme one and the operation was advised merely to give him a chance of improvement rather than offering him definite and permanent relief; the history of three and four convulsive seizures each night during a period of two years makes the prognosis most grave—no matter what the treatment. Up to the present time, however, the condition has certainly not progressed.

The right hemiparesis and paraphasia were probably due to a localized cerebral edema of the left cortex; it is noteworthy that this impairment has not returned since the left subtemporal decompression.

Case 122.—Old severe brain injury associated with a possible fracture of the base of the skull and with signs of an increased intracranial pressure; severe headache and convulsive seizures. Right subtemporal decompression. Temporary improvement only.

No. 574.—Joseph. Thirty-eight years. White. Married. Mechanic. U. S.

Admitted April 4, 1916—7 years after injury. Polyclinic Hospital. Referred by Doctor E. S. Bishop.

Operation April 14, 1916. Right subtemporal decompression and drainage.

Discharged May 2, 1916—18 days after operation.

Family history negative.

Personal History.—Always well and strong; of good habits and no alcoholism. Seven years ago while at work, patient was struck over the right side of the head by a large wooden beam; immediate loss of consciousness; profuse bloody and later a "watery" discharge from the right ear; taken to a hospital where he remained for 3 weeks, when he had so recovered that he was discharged as "well." During the next 4 years, he was able to work but not so vigorously as before the injury—complained almost daily of frontal and occipital headaches, "light-headed" spells upon stooping, and became easily fatigued. Two and a half years ago after a severe headache lasting 2 days, patient "fainted" momentarily but he did not fall to the ground; since that time, these "fainting" spells have occurred with increasing frequency so that, during the past 3 months, he has had as many as 11 attacks in one day; they occur chiefly during exertion and in the late afternoon—toward the end of a hard day's work. No major convulsive seizures have occurred at any time, although there is a history of having awakened 3 times in the morning to find that he had bitten his tongue and urinated involuntarily; headaches have become severe and he is now morose and melancholy; no longer takes an interest in current events; has been unable to work during the past 2 months. Patient has had competent medical treatment.

Examination upon admission (7 years after injury).—Temperature, 98.6°; pulse, 68; respiration, 18; blood-pressure, 144. Fairly well-developed and nourished; very much depressed and only answers questions upon insistence. No tremor of the tongue, lips or hands. No external evidence of former cranial injury. Hearing negative; otoscopic examination negative. No paralyses or impairments of sensation. Pupils equal and react normally. Reflexes—patellar very much exaggerated; exhaustible ankle clonus but no Babinski; abdominal reflexes both depressed but equal. Fundi—retinal veins dilated; nasal halves of both optic disks blurred by edema; no new tissue formation visible. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 15 mm.); Wassermann test negative and cell count was 7 cells per c.mm. X-ray (Doctor W. H. Stewart) —"no fracture of the skull."

Treatment.—Although no major convulsive seizures have occurred, the history of this patient, especially during the past 2 years and the increasing number of spells of the *petit mal* character, and the condition associated with definite signs of an increased intracranial pressure—these data would give the impression that the symptoms and signs of headache, dizziness and minor epileptiform spells were but the forerunners of convulsive seizures of the major type, and for this reason and in order, if possible, to prevent convulsions from occurring, the operation of right subtemporal decompression was advised—the patient, his parents and grandparents being right-handed and there being no localizing signs ascertained.

Operation (7 years after injury).—Right subtemporal decompression: usual vertical incision, bone removed, and no complications. Dura thickened, whitish, very vascular and under moderate tension; upon incising it, clear cerebrospinal fluid welled out, and throughout the operation the largest amount of cerebrospinal fluid escaped that I have yet seen; consequently the underlying cortex, owing to this rapid escape of cerebrospinal fluid, did not bulge and it pulsated normally. The cortex itself was very "wet" and edematous; several adhesions between the arachnoid and the overlying dura were severed. No gross pathological lesions visible—only a very "wet," edematous brain. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Uneventful operative recovery; operative inci healed *per primam;* patient did not complain of a "fainting" spell du his hospital convalescence and was discharged on the eighteenth day after operation. A second X-ray report—"bony defect of decompression operation with four silver clips demonstrated" (Fig. 145).

Examination (November 20, 1917—19 months after operation).—During the first 3 months after operation, patient was so improved that the headaches were lessened and the *petit mal* attacks so infrequent that it was hoped that he would be able to return to work; after working one week, the losses of consciousness returned, the headache became severe and the sense of early fatigue so great that the patient was obliged to give up work and has not worked since. In addition to the increasing frequency of the "fainting" spells, there has been a mental and emotional deterioration, so that now it is not possible for the patient to leave the house alone, and he has become so depressed and despondent that he sits for hours at a time by himself; he rarely speaks. For no reason apparently, tears will course down the patient's cheeks and even crying spells lasting over an hour occur; in many respects, the condition is similar to one of traumatic dementia. Decompression opening depressed and pulsates normally. Reflexes active but otherwise negative. Fundi—retinal veins possibly slightly enlarged; no edematous blurring of optic disk margins but both retinæ are rather suffused and edematous.

Last Report (September 20, 1918—29 months after operation).—Wife writes that patient is now in an institution as his condition gradually became worse so that he could not look after himself—even the most elementary things. No major convulsions, however, had occurred. Decompression area always remained depressed.

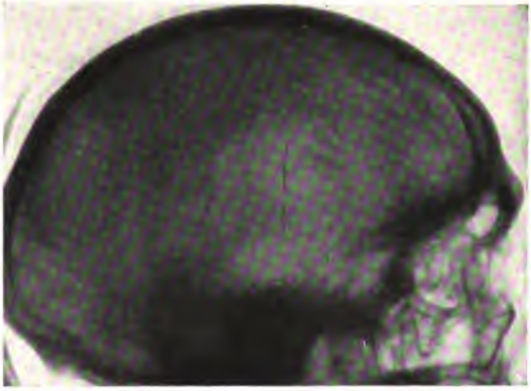

FIG. 145.—Oval bony defect of right subtemporal decompression in a patient following a fracture of the base of the skull and having signs of an increased intracranial pressure. Only temporary improvement following operation.

Remarks.—It would seem that this patient had developed most rapidly the advanced form of traumatic dementia and whether there was an underlying organic and constitutional basis for this marked mental and emotional deterioration cannot be stated; syphilis was not demonstrated as a factor nor were there ascertained any data of cardio-nephritic complications—in fact, no toxemic factor had been obtained. It is very surprising that no major convulsions have occurred in this patient, both on account of the history of the minor epileptiform spells of the *petit mal* type and on account of the pathology of the cerebral cortex as revealed at operation. The post-mortem findings will be most important in ascertaining the other causative factor in this patient's condition, if there is one; the permission for an autopsy has been obtained in writing.

E. Chronic Brain Injuries, not Associated with a Fracture of the Skull, with the Symptoms and Signs Persisting and Due to an Increased Intracranial Pressure.

If there had been no increase of the intracranial pressure ascertained in this series of patients, then a number of them would have been considered either as conditions of post-traumatic neurosis (a diagnosis so common and so tempting to make) or as the result of cerebral lacerations and contusions, and thus irreparable. The presence of the increased intracranial pressure, however, immediately made these patients amenable at least to treatment—either medical, or if this method failed, then surgical treatment—and thus it was possible for them to be benefited. It is only these selected patients having an increased intracranial pressure who can be consistently improved.

E. *Old brain injuries not associated with a fracture of the skull but with signs of increased intracranial pressure; symptoms and signs persisting. Subtemporal decompression. Improvement.*

Case 123.—Old severe brain injury not associated with a fracture of the skull but with signs of an increased intracranial pressure; severe headaches and emotional disturbances. Right subtemporal decompression. Recovery.

No. 343.—Barbara. Thirty years. White. Married. Housework. Russia.

Admitted August 20, 1915—1 year after injury. Polyclinic Hospital. Referred by Doctor O. S. Wightman.

Operation September 3, 1915. Right subtemporal decompression and drainage.

Discharged September 17, 1915—14 days after operation.

Family history negative.

Personal History.—Always well and strong. One year ago, patient was struck over the head by a heavy club while being attacked by a burglar; immediate loss of consciousness; no bleeding from either ear; taken in an ambulance to a hospital, where she remained 2 weeks. Since discharge, patient has had a continuous dull headache with severe exacerbations; she has never "fainted" nor has a convulsive seizure occurred. During the past 3 months, the headaches have become so severe and associated with vomiting spells that the patient has been obliged to remain in bed. Competent medical treatment has been unable to relieve the headache which at first had been considered as being a post-traumatic neurosis and entirely neurasthenic in character. Patient has become very irritable and depressed.

Examination upon admission (1 year after injury).—Temperature 98.8° pulse 70; respiration, 18; blood-pressure, 126. Rather poorly developed and nourished; very much depressed and melancholic. No external evidence of head injury. Hearing negative; otoscopic examination negative. No paralysis or impairments of sensation. Pupils equal and react normally. No nystagmus. Reflexes—patellar very much increased but equal; no ankle clonus nor Babinski; abdominal reflexes depressed but equal. Fu —retinal veins rather full; nasal margins of both optic disks blurred edema but otherwise negative. Lumbar puncture—clear cerebrospinal under increased pressure (approximately 14 mm.); first Wassermann 1 plus; second and third Wassermann tests negative; cell count was 7 per c.mm. X-ray (Doctor A. J. Quimby)—"no fracture of skull to be observed."

Treatment.—In the belief that the severe headache was due to a chronic cerebral edema following the brain injury of 12 months ago and since competent medical treatment was unable to relieve the condition and there being present definite signs of an increased intracranial pressure, a right subtemporal decompression was advised not only to relieve the headache, but also thereby to lessen the emotional instability and to prevent the danger of future complications—particularly epileptiform seizures. (No doubt, there is a large neurasthenic and neurotic factor in this case, but I believe it is superimposed upon and due to the definite organic lesion of chronic cerebral edema.)

Operation (12 months after injury).—Right subtemporal decompression: usual vertical incision and bone removed; while rongeuring away the bone, the middle meningeal artery was torn and much difficulty encountered in checking the rapid escape of blood; it was finally stopped by the application of a silver clip, low down, at the inferior border of the decompression opening. Dura slightly thickened and tense; upon incising it, clear cerebrospinal fluid oozed out, revealing a very "wet," edematous cortex under increased pressure. The rapid escape of cerebrospinal fluid permitted the cortex to recede and pulsate normally before the end of the operation. No gross lesion visible and only a "wet," swollen edematous cortex exposed. Usual closure with 2 drains of rubber tissue inserted. Duration, 90 minutes.

Post-operative Notes.—Uneventful operative recovery; within a week, the patient complained less of headache and at discharge on the fourteenth day post-operative, patient felt better than she had for months in that the headaches were not severe and she was not so depressed; incision healed *per primam.*

Examination (November 22, 1917—26 months after operation).—Patient has been practically well since the operation; during the past year, a dull headache occurred not more than once a month; she is no longer irritable and depressed, and in every way "seem to be myself again." Decompression area slightly depressed beneath the flush of scalp; normal pulsation. Reflexes active but otherwise negative. Fundi—retinal veins possibly slightly enlarged; no blurring of margins of optic disks and all details are clear and distinct.

Last Report (December 14, 1918—39 months after operation).—Patient writes, "I am as well as ever; nothing bothers me now outside of an infrequent headache but it's not much. Scar of operation sunken and I can feel it beating."

Remarks.—From the history of this patient, and if careful ophthalmoscopic and lumbar puncture examinations had not been made, it would have been very easy to have considered the patient as neurotic and neurasthenic—a condition of post-traumatic neurosis alone; and if there were no signs of an increased intracranial pressure, this diagnosis of a functional condition would have been the more probable one. But it having been ascertained that there was an increased intracranial pressure, then the diagnosis of simple neurosis is not sufficient and unless medical treatment is able to lower this increased pressure (usually due to a chronic cerebral édema following the injury), the condition cannot be improved by the usual treatment of functional conditions, such as psychotherapy, etc. When the usual medical means do not suffice in lowering the increased intracranial pressure of these patients, then the mechanical relief of the pressure is necessary by means of a subtemporal decompression—not only to improve the present condition but also to prevent the even more serious complications, such as convulsive seizures, marked mental and emotional impairments, etc.

The X-ray picture in not disclosing a fracture of the skull does not mean that a fracture of the skull had not been present; unless the X-ray picture is taken at the proper angle to disclose the fracture (if present), then its shadow will not be seen and a picture at a different angle will frequently reveal its presence. However, as has been stated before, the fracture of the skull (unless it is a depressed fracture of the vault), is possibly the most unimportant part of brain injuries, and frequently the most serious brain injuries are not associated with a "fracture of the skull," which is a term meaning little or nothing—except that the cranial injury was of sufficient force to fracture the skull and very frequently patients having this condition are not at all seriously injured in that the brain has not been damaged in the least, whereas only too frequently a severe brain injury results when no fracture has occurred.

CASE 124.—Old severe brain injury not associated with a fracture of the skull but with signs of an increased intracranial pressure; severe headaches, dizzy spells and emotional disturbances. Right subtemporal decompression. Recovery.

No. 855.—Mabel. Forty-five years. White. Married. Housewife. U. S.

Admitted May 5, 1915—15 months after injury. Polyclinic Hospital. Referred by Doctor C. H. Chetwood.

Operation May 16, 1915. Right subtemporal decompression and drainage.

Discharged May 30, 1915—14 days after operation.

Family history negative.

Personal History.—Always well and strong; no alcoholism. Fifteen months ago, patient was struck upon the head by the falling of a heavy trap-door; momentarily unconscious and remained in a semiconscious condition for 2 days; no bleeding from the ears; patient remained in bed at home for a period of 2 weeks, having severe headache and dizziness, which continued until 4 months ago, when the headache became so acute and the dizziness so extreme that the patient has been unable to walk any distance; marked increase of nervousness, so that the patient is very easily frightened; emotional upsets—crying spells and then very melancholic for several days at a time—always complaining of a severe headache; no "fainting" spells nor convulsive seizures at any time. Patient has had competent medical treatment.

Examination upon admission (15 months after injury).—Temperature 98.6° ; pulse, 78; respiration, 20; blood-pressure, 138. Fairly well-developed and nourished; very anxious expression and complaining of severe frontal and occipital headache. No external evidence of cranial injury. Hearing negative; otoscopic examination negative. No paralyses or impairments of sensation. Pupils equal and react normally. No nystagmus. Reflexes—patellar exaggerated but equal; no ankle clonus but suggestive double Babinski; abdominal reflexes obtained with difficulty. Fundi—retinal veins dilated; nasal halves of both optic disks obscured by edema—right possibly more than left. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 16 mm.); Wassermann test negative and cell count was 3 cells per c.mm. X-ray (Doctor A. J. Quimby)—"the picture is negative."

Treatment.—For fear that this patient might become so emotionally unstable that convulsive seizures would occur and there being a definite increase of the intracranial pressure as demonstrated by the ophthalmoscope and at lumbar puncture, and also since the routine treatment of general hygiene, diet, catharsis and the usual neurasthenic treatment of hydrotherapy, electrotherapy and psychotherapy have been of no benefit to the patient but the condition is becoming rapidly worse, it was decided to perform a right subtemporal decompression in the hope that the condition would be improved by thus lessening the increased intracranial pressure mechanically.

Operation (15 months after injury).—Right subtemporal decompression: usual vertical incision, bone removed, and no complications; bone was unusually thin and not vascular. Dura thin, transparent and bulging; upon incising it, clear cerebrospinal fluid spurted out under high tension, and upon enlarging dural opening the underlying "wet," edematous cortex tended to protrude but did not rupture, as the cerebrospinal fluid escaped rapidly in large quantity so that the cortex soon receded and pulsated normally. No gross cortical lesion demonstrated except the very "wet" and almost cystic condition of the pia-arachnoid. Usual closure with 2 drains of rubber tissue inserted. Duration, 35 minutes.

Post-operative Notes.—Uneventful operative recovery; incision healed *per primam;* patient complained of only dull headache and he was able to be discharged upon the fourteenth day post-operative.

Examination (October 4, 1916—17 months after operation).—Patient has made an excellent recovery; although she has a dull headache almost each week, it no longer interferes with her work and it is not associated with dizziness; "disposition" is much better in every way—is seldom depressed and only occasionally irritable. Husband says she is now "livable." Decompression area depressed beneath the flush of scalp and pulsates normally. Reflexes active but otherwise negative. Fundi—retinal veins of normal size; details of both optic disks clear and distinct.

Last Examination (September 4, 1918—40 months after operation).—The improvement of the patient has continued so that she is considered a "well woman"; she has only an occasional mild headache and if it were not on account of the fear that "my nervous fits" would return, patient says she would be perfectly happy; she is able to do her house-work. Decompression area depressed and pulsates normally. Reflexes active but otherwise negative. Fundi negative.

Remarks.—This patient has been a very interesting one to follow and to note the marked change—especially the lessening of the signs of pressure in the fundi. If it were not for the signs of the increased intracranial pressure, this patient would have been considered as suffering from a post-traumatic neurosis and that would have undoubtedly been her condition; however, upon ascertaining the signs of an increased intracranial pressure by means of the ophthalmoscope and at lumbar puncture, she was immediately removed from that large group of functional conditions, and when it was not relieved by the usual medical treatment, then it had to be treated mechanically—that is, the operation of subtemporal decompression.

CASE 125.—Chronic severe brain injury with a resulting cortical and supracortical hemorrhagic cyst formation directly beneath the site of the bullet-injury of the vault; no fracture of the skull. Mental retardation, emotional instability and Jacksonian convulsive seizures. No operation. Death from opium poisoning. Autopsy.

No. 014.—Ling. Thirty-four years. Yellow. Married. Clerk. China.

Admitted April 10, 1912—6 years after gunshot injury. St. Luke's Hospital, Shanghai. Referred by Doctor A. W. Tucker.

Died April 11, 1912—20 hours after admission.

Family history negative; both parents and the relatives were right-handed (as well as could be ascertained by questioning the relatives present). Patient has always been left-handed.

Personal History.—Always well and strong with the exception of the usual diseases of childhood. During the six years preceding the cranial injury, the patient worked as a clerk in the British-American Tobacco Company, where he was considered an unusually capable and intelligent employee. Seven years ago (November 6, 1905), the patient was shot in a Boxer uprising in the right temple; only momentary loss of consciousness; patient was able to walk to his home, and with the exception of a dull headache there were no complaints; the small scalp wound of bullet entrance was bandaged and the wound healed without the formation of pus. (At the time, it was not thought that a foreign body had entered the scalp wound.) Patient was able to return to his office work upon the following day, but on account of continuous severe headache and an inability to perform his work properly, he was obliged to give up his position after a period of ten days. He gradually became restless, irritable, unable to sleep well and complained of severe headache for a period of several months; no speech impairment, however, was observed at any time. The patient no longer appeared interested in things and would sit by himself for hours at a time, holding his head in both hands; if disturbed, he would become enraged, and on two occasions he assaulted his annoyers. Five months after the cranial injury, the first convulsive seizure occurred—beginning on the left side of the face, then the left arm, later the left leg and finally the entire body—and it lasted for over five minutes; apparently an entire loss of consciousness was present. During the past six years, the condition has gradually progressed in that the convulsive seizures (which always begin on the left side) have increased in frequency so that during the past three months, they have occurred as many times as six in one week; very irritable and excitable, and then, at times, very much depressed—crying for hours at a time on account of "pain in head"; no longer remembers the simplest things. Two months ago he drank a large amount of liquid opium with suicidal intent, but the vomiting was so profuse that he recovered; he stated at that time that he would attempt it again later.

Present Illness.—Thirteen hours before admission to the hospital, the patient swallowed a large quantity of liquid opium—he was profoundly unconscious; brought to the hospital upon a stretcher.

Examination upon admission (7 years after cranial gunshot injury and 13 hours after drinking the opium).—Temperature, 97°; pulse, 18 and very irregular; respiration, 6, and very shallow and irregular. Profoundly unconscious; cold, clammy skin. At times, the pulse and respiration practically cease—requiring artificial respiration. Both pupils are of pin-point size and non-reactive to light. Reflexes—both superficial and deep are all abolished. During the examination, a brother told the interpreter about the former gunshot injury, and upon careful palpation it was possible to feel a small foreign body directly beneath the old scar over the posterior portion of the right half of the frontal bone.

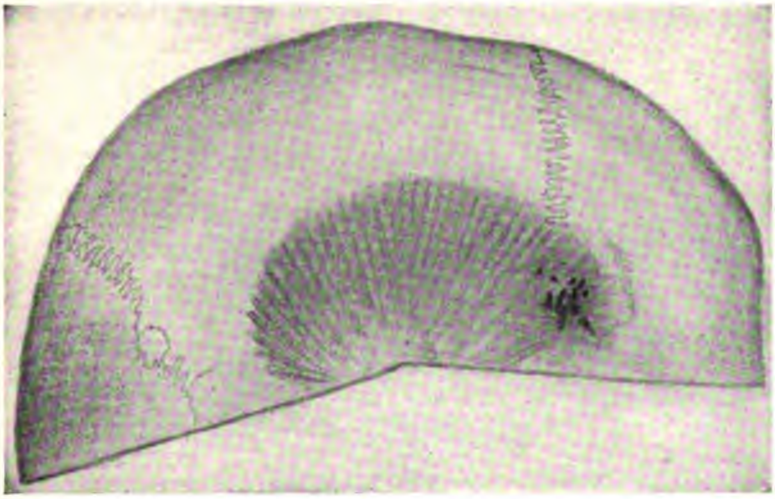

Fig. 146.—Small fragments of leaden bullet imbedded in the posterior portion of the right half of the frontal bone just anterior to the coronal suture. The outer table of the underlying bone was merely indented but very slightly. The attachment of the right temporal muscle to the parietal crest is well portrayed.

Treatment.—The usual emergency treatment for these patients—artificial respiration, vigorous massage and passive exercises, heated blankets and hot water bottles, repeated gastric lavage and high colonic irrigation, hypodermic injections of caffeine, etc. The condition of the patient, however, rapidly became worse in that the pulse and respiration became more and more irregular and also weaker, the pupils gradually dilated (permitting a careful ophthalmoscopic examination to be made) and the patient died 16 hours after admission and 29 hours after the ingestion of the opium. The fundi presented the appearance of a mild secondary optic atrophy with irregular nasal margins obscured by edema.

Autopsy of head alone: directly beneath the scar of the scalp from the former head injury in the right posterior frontal area, was a leaden bullet (fragmented) indenting slightly the outer table of the underlying bone (Fig. 146). No fracture of the bone could be found and no depression of the inner table of the vault was ascertained. The dura beneath this point of contact was definitely thickened over an area of 2 inches in diameter, and lying directly under it and connected with it by numerous adhesions was an oval supracortical and cortical hemorrhagic cystic formation of 3 cm. in diameter; it extended into the cortex to a depth of 2 cm. and occupied the posterior portion of the right second and third frontal convolutions—just anterior to the right fissure of Sylvius (Fig. 147); upon incising its outer wall, a straw-colored fluid escaped, permitting the cyst to collapse. The supracortical vessels adjacent to this cyst formation were indurated and

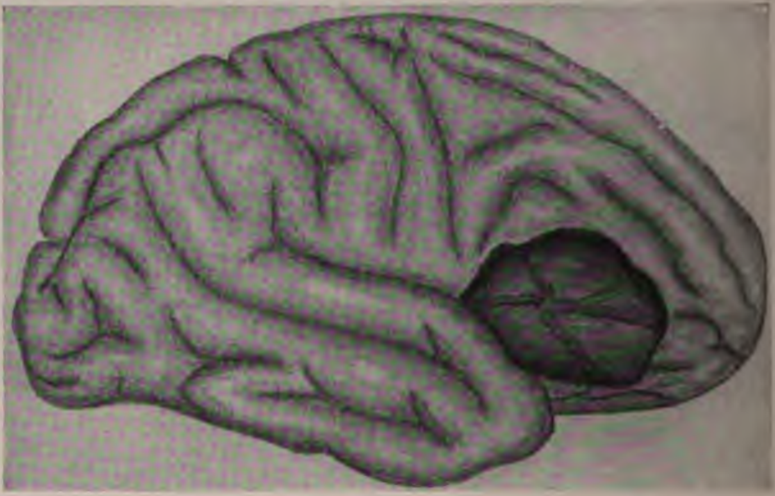

FIG. 147.—Dark hemorrhagic cyst in the posterior portion of the right frontal lobe and directly beneath the gun-shot injury of the vault—and no fracture of the bone present.

surrounded in the sulci by a cloudy thickening of connective tissue—the end-result of a former supracortical hemorrhage. The edematous cerebral cortex, other than that of the right frontal and the contiguous areas of the right temporo-sphenoidal lobes, was normal; no cortical adhesions found elsewhere. Ventricles were negative.

Remarks.—There can be no question that the cerebral injury occurring in this patient in good health was the primary cause of the increased intracranial pressure producing the headache and the mild secondary optic atrophy, and the later development of the mental and emotional impairment and the Jacksonian epilepsy. If a careful neurological examination had been possible within a short time after the injury, it is possible that this patient could have been permanently relieved and benefited by a right subtemporal decompression and drainage of the supracortical and cortical hemorrhage; at least, the increased intracranial pressure could have been lowered and thus the headache avoided and the impairment of vision prevented—as well as lessening the danger of the later mental and emotional impairment and the convulsive seizures. It is most doubtful if a marked improvement could have been obtained after the cortical irritability had been so increased that the convulsive seizures were occurring in such great frequency—the so-called epileptic habit; it would, however, have been advisable to perform the operation even at this late date in the hope that an improvement was possible—at least a retardation of the progress of the deterioration.

The absence of a fracture of the vault of the skull in this patient is interesting. The situation of the hemorrhagic cyst in the second and third right frontal convolutions posteriorly in a left-handed patient and yet no motor impairment of speech is most suggestive; the fact that his relatives and ancestors were all right-handed and therefore their motor speech centers were presumably in the cerebral cortex of the left frontal lobe, and that in this left-handed patient a destruction of his theoretical motor speech centre in the cortex of the right frontal lobe did not produce a speech impairment, would tend to indicate that in this patient at least, the motor speech area of the cerebral cortex is situated in either cerebral hemisphere more as the result of one's ancestry and heredity rather than as influenced by the individual himself, whether right-handed or left-handed; in this patient naturally, one would expect the motor speech area to be in the right cerebral cortex—the patient himself being left-handed, but the pathology as disclosed by the autopsy would tend to confirm the belief that in this patient, at least, the motor speech area was in the usual posterior portion of the left third frontal convolution (Broca's area).

F. Chronic Brain Injuries Complicated by Other Conditions

Among the more common complications occurring in the patients having chronic brain injuries are cardio-vascular and cardio-renal diseases, with and without the factor of alcoholism; brain tumors which frequently appear beneath and at the site of the former cranial and cerebral lesion; the various manifestations of lues, and especially of the cerebrospinal type and of paresis itself; mental derangements, and particularly the frank forms of traumatic dementia and the other types of mental disease which occasionally follow severe brain injuries, as though the intracranial lesion had at least precipitated the mental impairment; it is only in the exceptional patient that the history of the cranial trauma is the direct cause for the permanent mental derangement; the cerebral lesion may be a predisposing cause but rarely, if ever, the sole factor in the condition of frank manic-depressive insanity, dementia præcox and even of traumatic dementia itself.

A. *Nephritis.*

Case 126.—Chronic severe brain injury associated with a linear fracture of the vault, high intracranial pressure and with nephritis. Right subtemporal decompression; improvement. Spontaneous hemorrhage later into left ventricle; death. Autopsy.

No. 583.—William. Twenty-seven years. White. Single. Electrician. United States.

Admitted May 25, 1916—7 months after injury. Polyclinic Hospital. Referred by Doctor J. F. White.

Operation June 12, 1916—17 days after admission. Right subtemporal decompression.

Died September 4, 1916—11 months after injury and 82 days after operation.

Family history negative.

Personal History.—Perfectly well until 9 months ago (2 months before injury), when patient had severe headaches each morning upon arising and lasting over 2 hours; no frequency of urination and no medical attention was requested. Seven months ago while at work, patient was struck upon the head by a large iron bolt; no loss of consciousness; watery fluid, however, escaped from the right ear and continued for 3 days after the injury, when the headache again returned. Patient continued working during the following month until the general headache became so severe that he was obliged to remain in bed; no other complaint except persistent and continuous headache. Repeated urine examinations revealed much albumen and many hyaline and granular casts.

Examination upon admission (7 months after injury).—Temperature, 98.8°; pulse, 82; respiration, 26; blood-pressure, 200. Well-developed and nourished. Perfectly conscious but rather drowsy and complaining of severe frontal headache. Impairment of hearing of right ear—bone conduction being greater than air conduction; otoscopic examination reveals a small irregular perforation in the posterior half of right tympanic membrane. Pupils equal and react normally. Reflexes—patellar very much exaggerated—right more than left; right ankle clonus inexhaustible—left exaustible; right Babinski and plantar flexion not obtained on the left foot; abdominal reflexes—right absent. Fundi (Doctor J. A. Kearney)—"regenerative choked disks; entire outline of left disk was obliterated, although no tissue elevation; right—no degeneration noticed but disk outlines are obscured in old exudate. Both fundi are congested and suffused; retinal veins enlarged." Lumbar puncture—clear cerebrospinal fluid under high pressure (approximately 22 mm.); Wassermann test negative and cell count was 4 cells per c.mm. X-ray (Doctor W. H. Stewart)—"small linear fracture extending obliquely through the lower posterior part of the right parietal bone toward right frontal area" (Fig. 148). Urine examination (Doctor W. L. McFarland)—"heavy trace of albumen; numerous hyaline and finely granular casts."

Treatment.—Vigorous medical treatment was administered by Doctors Wightman and Bishop in the hope that the increased intracranial pressure, due to the cerebral edema associated with nephritis in a patient having the history of the cranial injury, could be lessened and thus the vision be spared. Within 2 weeks after admission, the condition steadily progressed so that the blood-pressure became 240, the ophthalmoscope revealed "choked disks" of 5 diopters and a lumbar puncture registered the pressure of the cerebrospinal fluid as being approximately 28 mm.; patient became more and more stuporous, so that it was now considered advisable to perform a right subtemporal decompression to lower the increased intracranial pressure mechanically and thus, at least, save the eyesight and permit the patient to withstand the nephritis much better than when subjected to this high intracranial pressure.

Operation (17 days after admission).—Right subtemporal decompression: usual vertical incision, bone removed, and no complications; much bleeding occurred, however, due to the high blood-pressure. Dura exceedingly tense, and upon incising it, a large quantity of cerebrospinal fluid escaped in gushes as though walled off by adhesions; the underlying "wet," edematous cortex protruded but did not rupture, and owing to the rapid escape of much cerebrospinal fluid, the brain pulsated at the end of the operation. No cortical hemorrhages or lacerations visible—merely a very "wet" brain under high pressure. Usual closure with 2 drains of rubber tissue inserted. Duration, 55 minutes.

Post-operative Notes.—Patient made an excellent operative recovery so that at the end of 22 days he was sent home under medical treatment.

Examination at discharge (22 days after operation).—Temperature, 98.6°; pulse, 80; respiration, 24; blood-pressure, 178. No complaints except slight dull headache in the morning. Decompression area bulges slightly and pulsates normally. Pupils equal and react normally. Reflexes—patellar exaggerated but equal; double exhaustible ankle clonus but suggestive right Babinski; abdominal reflexes present and equal. Fundi—retinal veins enlarged; both fundi congested and suffused but only nasal halves of optic disks blurred by edema; both physiological cups shallow and filled with new tissue and the margins of optic disks rather irregular. Urine examination—heavy trace of albumen and numerous hyaline and finely granular casts.

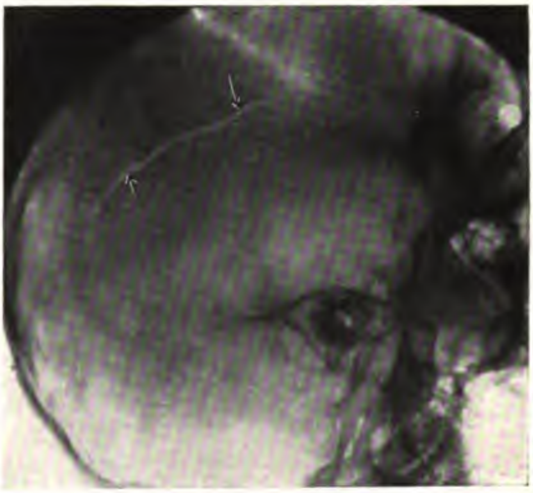

FIG. 148.—Small linear fracture of right parietal bone in a patient having chronic nephritis and a high intracranial pressure producing a secondary optic atrophy. Death due to later spontaneous hemorrhage into left ventricle.

Treatment.—Patient was carefully instructed regarding his medical treatment, diet, etc., and was advised to return each week to the out-door medical clinic for observation.

Examination (August 22, 1916—48 days after discharge).—Patient returned to the hospital for the first time and complained of intense frontal headaches, nausea and frequent vomiting—the symptoms and signs of the toxemia of nephritis. Temperature, 99°; pulse, 88; respiration, 26; blood-pressure, 204. Decompression area bulging tensely. Pupils equal and react normally. Reflexes—patellar very active but equal; double ankle clonus and double suggestive Babinski; abdominal reflexes obtained with difficulty. Fundi—double "choked disks" of 6 diopters; retinal veins dilated and buried in the edematous tissue of retinæ. Urine exmaination—large amount of albumen and many hyaline and granular casts.

Treatment.—Patient was admitted to the ward and the most vigorous medical treatment administered, so that within one week patient was apparently in much better general physical condition and the headaches had almost entirely disappeared. A röntgenogram was negative except for the bony defect of the decompression opening; three silver clips are shown. On August 29 (7 days after admission), when patient seemed to be making an excellent recovery, a left hemiplegia suddenly occurred and the following examination was made: Temperature, 99°; pulse, 104; respiration, 30; blood-pressure, 230. Profoundly unconscious and in state of collapse. Decompression area was "tight as a drum" and no pulsation palpable. Pupils—left dilated and does not react to light. Reflexes—patellar exaggerated, left being greater than right; double ankle clonus and double Babinski; abdominal reflexes—left absent, right depressed. Fundi—retinal veins dilated; "choked disks" of 6 diopters. Lumbar puncture—clear cerebrospinal fluid under exceedingly high pressure (approximately 30 mm.). Urine examination—large amount of albumen and many hyaline granular casts.

Treatment.—In spite of vigorous medical treatment, the condition of patient rapidly became worse, so that 12 hours after the hemiplegic attack the temperature ascended to 106°, pulse to 160 plus, and respiration to 48, while the blood-pressure now descended to 116; pulmonary edema appeared and patient died—16 hours after hemiplegic attack and 82 days after operation.

Autopsy (Doctor T. D. Lehane): over the posterior portion of right parietal area was a small depressed fracture of the outer table of the vault alone; one inch below this area was a linear fracture extending obliquely forward into right frontal bone; small fracture of right petrous bone. No subdural hemorrhage present. In the left lateral ventricle was a tense hemorrhagic clot, the size of a lemon, and thus compressing directly the left pyramidal tract fibres. Right lateral ventricle negative. Brain itself was very edematous and swollen. Subtentorial region negative.

Remarks.—Having thus been enabled to trace this patient from almost the beginning of his condition to the end, we are in a position to estimate the various factors complicating the condition, and it would appear that a chronic nephritis was the underlying cause of the intracranial condition which was precipitated by the cranial injury; that is, the cranial injury caused an acute cerebral edema to occur chiefly on account of the prese
the chronic nephritis, and there may have been already a mild cerebr
even at the time of the cranial injury and due to the chronic
alone—the presence of headache for the two months preceding
injury might indicate this.

It is interesting to observe the immediate improvemen
condition and particularly the lessening of the intrac

result of the subtemporal decompression; naturally, it was advised only as an immediate means of sparing the vision and to enable the patient to resist a nephritis more effectively; it was the first patient that I had ever advised the operation of subtemporal decompression to lessen an increased intracranial pressure primarily due to a chronic nephritis, even though complicated by a cranial injury, and it was only advised after all medical treatment had failed to retard the progress of the condition. The temporary improvement following the operation was most encouraging.

The sudden left hemiplegia and the autopsy findings of a large left ventricular hemorrhage are difficult to correlate; anatomically, it would seem that the left pyramidal tract fibres should have been more compressed than the right pyramidal fibres, and I am at a loss to explain it satisfactorily—unless the indirect pressure of a clot in the left hemisphere could exert a greater compressive effect on the pyramidal tract fibres of the right hemisphere than upon those of the left hemisphere—and this does not seem logical. Careful examination of the right hemisphere and the right pyramidal tract fibres down through the internal capsule did not reveal any lesion.

It is interesting to note in the history that the patient had had severe headache during the two months preceding the injury and then, in spite of the cranial injury sufficient to cause a fracture of the skull, this patient did not complain of headache during the 3 days following the injury and while cerebrospinal fluid was escaping through the line of fracture out of the right ear; that is, this discharge of cerebrospinal fluid so lessened the increased intracranial pressure of the chronic cerebral edema due to the pre-existing nephritis, that the headache disappeared and only returned when the cessation of the flow of cerebrospinal fluid from the ear occurred—3 days after the cranial injury. This is an excellent illustration of "natural decompression" afforded the patient by the fracture of the skull; fortunately, no infection through this line of fracture into the ear occurred—the longer the aural discharge persists, the greater the danger of infection and a resulting meningitis.

The presence of the "regenerative choked disks" indicates an increased intracranial pressure of long duration, and in this patient its mild secondary optic atrophy was exhibited and therefore a permanent impairment of the vision of mild degree would result.

Following the sudden left hemiplegia with extreme intracranial pressure, the right decompression area and right ventricle might naturally have been explored with negative results.

B. *Tumor of the brain.*

Case 126a.—Old cranial injury associated with mild signs of an increased intracranial pressure; osteo-sarcoma of left squamous bone; subcortical cerebral sarcoma. Operation. Improved.

No. 260.—Linley. Twenty-nine years. White. Married. Physician. U. S.

Admitted May 26, 1915—5 years after second injury. Polyclinic Hospital. Referred by Doctor C. C. Sweet.

First operation July 13, 1915—47 days after admission. Left subtemporal decompression and partial craniectomy.

Discharged August 24, 1915—41 days after operation.

Second operation March 12, 1916—8 months after first operation. Left exploratory osteoplastic operation.

Discharged April 6, 1916—24 days after operation.

Family history negative.

Personal History.—Patient was an only child, full term, normal delivery and considered a normal boy; usual childhood diseases. When one year of age, patient fell from a chair, striking the left side of head against the leg of the table; unconscious for 15 minutes; several drops of blood trickled from left external auditory canal; after 10 days in bed, the condition was excellent and the injury was forgotten. As a boy, patient had severe frontal headaches twice each month. In 1910 (5 years before admission), patient fell from a motor-cycle, striking the left side of head; no loss of consciousness but dazed for a number of minutes. Patient considered himself perfectly well until February, 1911 (4 years ago), when he suddenly became aware of a numbness beginning in the right hand and extending over the entire right half of the body—a "sensory convulsion" and no motor signs; this attack lasted one minute, and then the sensation became normal. These sensory spells occurred daily several times—always beginning in the right hand and limited entirely to the right side of the body—and they have continued up to the present time; during 1913, there was an interval of 3 months during which time no sensory spells occurred. In January, 1914 (17 months before admission), patient had his first Jacksonian convulsive seizure: following the sensory spell, the fingers of the right hand twitched, then the entire right arm, and finally the entire right half of the body—and no loss of consciousness; each spell lasted from 2 to 3 minutes. On May 1, 1914 (12 months ago), patient had his first convulsive seizure associated with loss of consciousness; the attack began as usual in a numbness of the right side of the body, then a motor convulsion of the right arm and then the right side of face and right leg, which now became a general convulsion when the patient lost consciousness; this attack lasted 15 minutes and he dislocated both arms at the shoulder during it. These major seizures of this character and type have continued every few days, while the minor attacks of numbness of the right side of the body and slight Jacksonian seizures limited to the right arm have been daily, and frequently several times each day. During this period, patient has had severe frontal and occipital headaches lasting 24 hours at a time. During past year he has been obliged to wear hats three sizes larger than before.

Present Illness.—Patient now comes to the hospital complaining not only of the numbness of the right side of the body, which is always present, and of the daily Jacksonian convulsions and of the frequent general convulsions, but a definite motor aphasia (incomplete) and paraphasia have occurred associated with an inability to "concentrate my mind" and a distinct loss of memory, especially for recent events.

Examination upon admission (years after injury).—Temperature, 98.8°; pulse, 80; respiration, 24; blood-pressure, 134. A fair development and nourishment. Perfectly rational and conscious, but a distinct hesitancy in speech and a difficulty in using proper words; no sensory aphasia. No external evidence of cranial lesion. Hearing negative; taste and smell negative.

No nystagmus. Romberg test negative. Pupils equal and react to light normally. Reflexes—patellar active, right more than left; right exhaustible ankle clonus and tendency to right Babinski; abdominal reflexes—right depressed. Fundi—retinal veins enlarged; upper and lower nasal quadrants of both optic disks slightly blurred—left possibly more than right; physiological cups rather shallow, but no "choked disks" nor measurable swelling of the disks. Lumbar puncture—clear cerebrospinal fluid under slightly increased pressure (approximately 13 mm.); Wassermann test negative and cell count was 5 per c.mm. X-ray (Doctor A. J. Quimby)—"an irregular line of fracture extending horizontally through the left squamous bone backward to the left occipital bone; in this area there is a definite blur, possibly of thickened bone." Urine examination negative. Definite weakness of the right arm, but no weakness of right leg or right side of face can be elicited. Careful sensory tests of right side of the body reveal no sensory impairment to light-touch, pain, or temperature; no astereognosis nor apraxia.

Treatment.—After studying this patient and examining him frequently during a period of 45 days in which a number of major convulsions were accurately reported, it was finally decided that a left subtemporal decompression and exploration should be performed. (Owing to the fact that this patient had been repeatedly assured that his condition was due to intestinal stasis and the operation of colectomy advised as a remedy for his ills, it was with great difficulty that the patient could be convinced that there was a definite local lesion underlying the left side of his skull which was the possible cause of his condition.)

First Operation (47 days after admission).—Left subtemporal decompression, exploration and removal of tumor of bone: usual vertical incision, bone removed, and no complications; extending transversely through the lower portion of the left squamous bone was an irregular line of an old fracture; the bone was 2 cm. in thickness and very hard, extending inward and depressing the underlying dura; this thickened area of bone formed an irregular prominence of 2 inches in width; it was entirely removed. The underlying dura was thickened, whitish and tense, and upon incising it, a very edematous "wet" swollen cortex tended to protrude but did not rupture; upon the cortex, especially in the sulci and about the cortical veins, there was a hazy whitish induration—the result of a former subarachnoid hemorrhage at the time of the cranial injury. It was considered that sufficient cause extradurally had been found to account for the patient's symptoms and signs, and as the operation had already lasted 90 minutes and the patient not being in good condition due to much pulmonary mucus resulting from the ether, and on account of the loss of much blood due to the removal of a large area of thickened bone, it was, therefore, decided not to explore the brain itself. Usual closure with 2 drains of rubber tissue inserted.

Post-operative Notes.—Patient had a very stormy convalescence; the bronchitis following operation developed into a right pneumonia and then pleurisy, but after these complications the patient made an excellent recovery eventually and was able to leave the hospital 41 days after operation.

Examination at discharge (41 days after operation).—Temperature, 98.6°; pulse, 82; respiration, 24; blood-pressure, 130. No complaints of headache and patient has not had a minor spell or major seizure since the operation. Slight weakness of right arm persists but no impairment of sensation can be elicited. Decompression area bulges slightly beyond flush of scalp; pulsation normal. Pupils equal and react normally. Reflexes—patellar active, right possibly more than left; no ankle clonus and no Babinski; abdominal reflexes—right less active than left. Fundi—retinal veins slightly enlarged; nasal margins of both optic disks indistinct but other details of optic disks clear. A second X-ray disclosed the left decompression opening, the posterior portion of the line of fracture and three silver clips *in situ* (Fig. 149).

After the patient returned home, the first minor spell (entirely sensory in character) occurred 13 weeks after operation and continued at irregular intervals of 2 to 3 times a week. The first major seizure occurred on October 15, 1915—92 days after operation; the second major seizure following operation occurred 3 weeks later, and then both minor spells and major seizures reappeared with increasing frequency until the patient returned to the hospital in March, 1916.

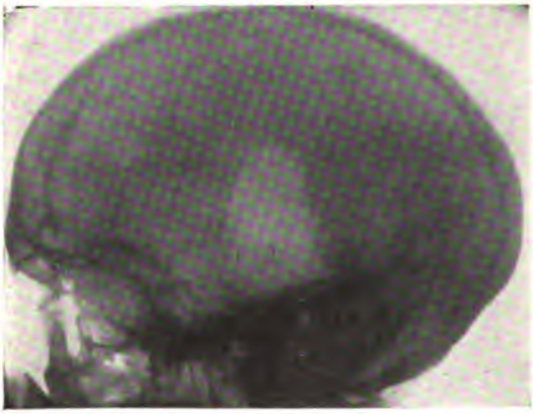

FIG. 149.—Oval bony defect of left subtemporal decompression in a patient having convulsive seizures following an old fracture of the left vault, and associated with an increased intracranial pressure; marked improvement following its operative lowering and removal of osteo-sarcoma of left squamous bone.

Examination upon second admission (8 months after first operation).—Temperature, 98.6°; pulse, 70; respiration, 18; blood-pressure, 140. Conscious. Decompression area flush with surrounding scalp; normal pulsation. Increasing difficulty of speech—chiefly a paraphasia; no sensory aphasia. Unable to recall recent events. Complains of continuous numbness of right side of body; no sensory impairment can be elicited and no astereognosis nor apraxia. No impairment of taste, smell or hearing. No dreamy states of unreality nor temporo-sphenoidal "fits." No limitation of the visual fields and no hemianopsia. Pupils equal and react normally. Reflexes—patellar exaggerated, right more than left; double exhaustible ankle clonus and suggestive right Babinski; abdominal reflexes—right depressed. Fundi: retinal veins enlarged; slight blurring of nasal margins of both optic disks. Definite weakness of entire right side of body, however—arm more than leg; right facial weakness of cortical type (right forehead mus not involved). No impairment of sensation can be elicited to light-touc pain or to temperature.

Treatment.—As the condition had only been temporarily improved the last operation and as the neurological examination still indicate lesion of the left hemisphere, it was considered advisable to perform a left osteoplastic flap operation and exploration of the left hemisphere.

Second Operation (8 months after first operation).—Left exploratory osteoplastic operation: usual curvilinear incision over left lower parietal area; bone-flap "turned down" and no complications. Dura incised in curvilinear manner; underlying cortex tense and in sulci about the vessels was a whitish cloudy induration due to the former subarachnoid hemorrhage at time of cranial injury. Upon attempting to tap the ventricle with a puncture needle, a rather resistant mass was encountered 3 cm. beneath the cortex and underlying the lower portion of left Rolandic area; this tumor mass was apparently the size of an orange. (Small specimen removed for examination was reported by Doctor Jeffries as being "small-celled sarcoma.") It was considered better surgical judgment not to attempt a removal of this tumor on account of its position beneath the left motor cortex and thus a more or less complete paralysis of the right side of the body would have resulted from its operative removal, and also the fact that it was of soft consistency and therefore undoubtedly malignant, and thus a recurrence would be most liable even if the tumor could now be surgically removed; again, it was hoped that the tumor might extrude itself gradually through the upper portion of the temporal lobe toward the site of decompression and thus its removal might be effected with very little damage (particularly is this so with spinal cord tumors of the medullary type). Usual closure and bone-flap replaced. Duration, 85 minutes.

Post-operative Notes.—Patient made an excellent operative recovery and he was discharged 24 days after operation.

During the past 30 months, I have repeatedly examined this patient. Much to our surprise, this patient was able to return to his work, which he continued for 9 months after operation; then on account of an occasional major seizure, patient accepted an out-door position and was able to work until 4 months ago, when on account of increasing aphasia and the frequency of both minor spells and major seizures, the patient has been living quietly at home.

Last Examination (November 29, 1909—54 months after first operation and 44 months after second operation).—Patient complains of persistent numbness of right side of body, particularly right arm, weakness of right arm and frequent minor spells and major seizures always beginning on right side of body. No headache and no impairment of vision. No impairment of taste, smell or hearing. Decompression area bulges under tension; upon performing lumbar puncture, this protrusion disappeared entirely when 2 ounces of cerebrospinal fluid were removed. Pupils equal and react normally. Reflexes—patellar active, right more than left; exhaustible right ankle clonus but no Babinski; abdominal reflexes—right less active than left. Fundi—nasal margins of both optic disks clear; retinal veins enlarged. No objective sensory impairment of right side of body, and no astereognosis and no apraxia. Definite weakness of right arm and less so of right leg and right side of face. Upon removing cerebrospinal fluid at lumbar puncture, patient experienced a marked improvement during the following 6 weeks in that no major seizures occurred and his general condition improved in every way. As a result of this observation, a lumbar puncture and withdrawal of 2 ounces of cerebrospinal fluid is now being performed each week.

Remarks.—This is a most instructive case in that it would seem that a sarcoma of the vault had occurred years following a fracture of the skull in that area; the definite symptoms and signs indicating a lesion of the left hemisphere were, at operation, considered as being due to the tumor of the bony vault alone, and naturally the underlying cerebral cortex and subcortex were not explored, merely a left subtemporal decompression being performed; later, when the patient's symptoms and signs persisted, a second operation was performed and a subcortical sarcoma, the size of an orange, located; owing to its position and undoubted malignancy, its operative removal was not attempted in the hope that it might extrude itself toward the decompression opening. At the last examination, when the patient entered the hospital having a hernial protrusion at the site of the decompression opening, it was hoped that this extrusion of the tumor had indeed occurred, but upon removing the cerebrospinal fluid at lumbar puncture this protrusion disappeared entirely and thus it may be considered as being due merely to blocked cerebrospinal fluid. The patient's condition during the past 6 months has been steadily becoming worse, and it is only since the repeated withdrawal of cerebrospinal fluid at lumbar punctures that a marked improvement in this condition has occurred; undoubtedly, these improvements, however, are only temporary.

It was hoped at the time of the second operation that, if the tumor did not extrude that it might degenerate by the formation of cysts, similarly to the frequent cystic degeneration of gliomata and thus the malignancy of the tumor be lessened and that possibly an ultimate recovery might occur; this fortunate result is indeed of rare occurrence, but it is possible, although the usual length of life following the existence of cerebral tumors of malignant character is on the average only 3 years. It was most important in this case, as in all patients having brain tumors, that a decompression should be performed early, no matter whether the tumor is malignant or not; in this way, the headaches are lessened, the vision is spared and a fairly comfortable existence is assured to the patient; besides, if the tumor is not malignant, then its early localization and removal is possible, and yet the vision of the patient is spared, and if the tumor should be malignant then the patient can be assured a fairly comfortable existence for several years, and yet the patient be not blind and a pitiful sufferer from the severe continuous headache. There is also, fortunately, the possibility that the diagnosis of malignancy of the tumor is a mistaken one—and this mistake does occasionally occur and possibly more frequently in the diagnosis of tumors of the brain, so that, the increased intradural pressure having been relieved by the decompression and if necessary a bilateral decompression, then the patient may be able to lead a fairly comfortable life, if not to regain his former mental and physical activity; the vision will at least have been spared, the headaches stopped and in every way a marked improvement obtained—even if only a temporary one of several years.

Case 127.—Old severe brain injury associated with an increased intra-

cranial pressure and later with convulsions. Right osteoplastic exploration; improvement. Emotional changes later associated with increasing intracranial pressure. Left subtemporal decompression and exploration; supracortical angiomatous plexus formation. Improvement.

No. 829.—Stephen. Twenty-three years. White. Single. Student. U. S.

Admitted March 22, 1917—15 years after injury. Johnson-Willis Hospital, Richmond, Va. Referred by Doctor Beverly R. Tucker.

Operation April 4, 1917.—Left subtemporal decompression, exploration and drainage.

Discharged April 30, 1917—16 days after operation.

Family history negative.

Personal History.—Full term baby, difficult labor and no abnormalities observed immediately after birth. Usual childhood diseases. Patient was considered a normal boy until he was 8 years of age, when he began to have severe headaches and occasional "dizzy" spells and 12 years ago (when patient was 11 years of age), severe epileptiform convulsions occurred and of such frequency that the condition became one of status epilepticus—general convulsions with loss of consciousness and an attack occurring every 20 minutes. Patient was taken to the Johns Hopkins Hospital, where Doctor Harvey Cushing performed a right osteoplastic exploration and relief of the high intracranial pressure; a very "wet," edematous cortex under high tension was revealed; no other abnormality noted. Patient made an excellent operative recovery—quickly regained consciousness, convulsions subsided and did not recur. Markedly improved for one year, when slight dull headaches and occasional "dizzy" spells reappeared, but never so severe as before the operation. It was now noticed, however, that the patient was changing emotionally in that he became less affectionate than before, and would lie needlessly, showing a marked tendency to steal and to burn articles both of value and then again trifles. In the meantime, the patient had progressed through the various elementary and secondary schools and finally reached college, where he was considered a normal boy, although rather "eccentric." Patient had periods of "wanderlust," when he would make trips to no purpose—merely because he was restless and could not remain quiet. The desire to burn and steal larger things increased and he finally yielded to a frequent temptation by burning a large building and stealing numerous articles—some of value, others not; this last offence occurred 4 months before admission to the hospital.

Examination (in consultation with Doctors Tucker and Willis, April 6, 1917).—Temperature, 98.2°; pulse, 66; respiration, 18; blood-pressure, 132. Well-developed and nourished. Perfectly conscious but reticent and apparently cynical; emotional reactions depressed and upon superficial examination, the condition might have been considered an early case of dementia præcox; patient was apparently oblivious to his surroundings, not affectionate to his relatives and he did not seem to realize the notoriety and the humiliation of his family due to the immensity of his crime—there being a criminal charge against him. Area of the former left osteoplastic operation was negative. No impairment of taste, smell or hearing.

Pupils equal and react normally. Reflexes—patellar very active, right greater than left; no ankle clonus but suggestive right Babinski; abdominal reflexes—right depressed. Fundi—retinal veins enlarged; nasal margins of both optic disks blurred and lower nasal quadrant of left optic disk obscured by edema. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 16 mm.); Wassermann test negative and cell count was 8 cells per c.mm. X-ray (Doctor A. L. Gray)—"convolutional pressure markings upon the inner tables of the vault over the frontal area; new bone formation about the periphery of the former exploratory operation." No weakness of arms or legs; no sensory impairment; no aphasia or astereognosis.

Treatment.—On account of the increased intracranial pressure and the localizing signs of a greater involvement of the left hemisphere, a left subtemporal decompression and exploration was advised in the belief that a lowering of this increased intracranial pressure would lessen the emotional instability of the patient by diminishing the cortical irritability and thereby cause an improvement, and also diminish the danger of the occurrence of future convulsions; in brief, the operation was advised in the hope that it might give the patient a definite chance of recovery by lowering the increased intracranial pressure.

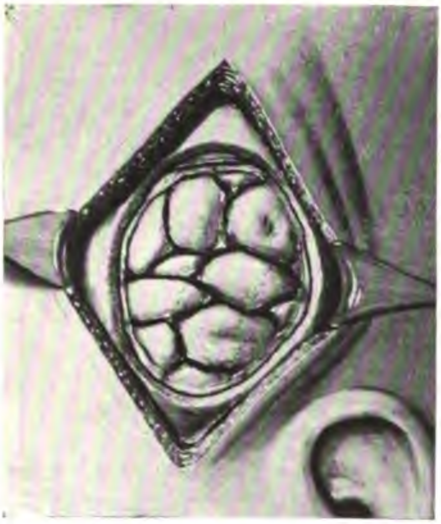

FIG. 149a.—The normal appearance of the cerebral cortex in the area exposed by the operation of left subtemporal decompression

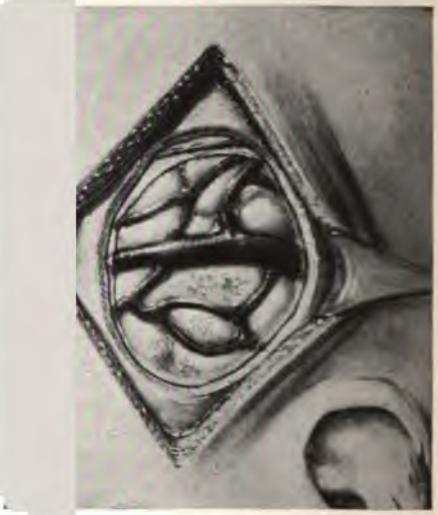

FIG. 149b.—Angiomatous plexus of enlarged supracortical veins associated with a high intracranial pressure, as disclosed by the left subtemporal decompression upon this patient.

Operation (15 years after injury).—Left subtemporal decompressio
exploration: usual vertical incision of 3½ inches in length extending
the left parietal crest down to the left zygomatic arch—½ inch ante
the left external auditory meatus. Skin flaps retracted and the te
fascia and muscle were now incised similarly in the direction of
cular fibres; these were retracted, exposing the underlying squa

tion of the temporal bone and the lower portion of the left parietal bone, which were now opened and rongeured away to a diameter of 2½ to 3 inches, revealing a tense thickened fibrous dura, non-transparent and under high tension, so that it tended to protrude. Upon making a small opening in the dura, clear cerebrospinal fluid spurted a distance of 1 to 1½ inches, and upon enlarging this opening to the diameter of 1 cm., the underlying arachnoid and cerebral cortex tended to protrude, indicating the high degree of intracranial pressure. This opening was quickly enlarged for fear a rupture of the cortex would occur by allowing the brain to be protruded under high pressure. Over the upper portion of the brain exposed was a mass of dilated blood-vessels some of them over ⅓ of an inch in diameter—forming an angiomatous plexus pressing down upon the cortex of the brain; the walls of these vessels were thickened, whitish, and at the upper portion was a bluish background as though this angiomatous mass was the result of a former supracortical hemorrhage which was becoming organized with new vessel formation. During the operation, a large amount of clear cerebrospinal fluid escaped and continued to drain; this loss of cerebrospinal fluid allowed the cerebral cortex to pulsate but it was still under increased pressure in spite of the large loss of cerebrospinal fluid; for this reason, 4 linen strands were inserted beneath the dura and brought out through the temporal muscle and temporal fascia, beneath the scalp, so that this increased amount of cerebrospinal fluid would be permanently drained by these artificial channels of drainage—as is performed in the operation for external hydrocephalus. No attempt was made to remove the angiomatous formation lying upon the surface of the brain for fear of hemorrhage; the overlying bone was removed and this permanent relief of the intracranial pressure, both by the removal of the bone and by the permanent drainage of the increased amount of the cerebrospinal fluid, should cause a definite improvement of the patient's condition, mentally as well as physically; the impairment of vision should also be improved. The wound was closed in the usual manner by bringing the temporal muscle and fascia together, and suturing them and the scalp in layers with catgut and finally silk.

Post-operative Notes.—Two hours after operation, the patient was regaining consciousness and apparently in excellent condition. Patient made an excellent operative recovery, so that he was discharged 16 days after operation; incision healed *per primam*. This patient has been repeatedly examined during the past 18 months and his entire condition, especially his emotional reactions, have been very much improved; he is no longer gloomy and morose, he is interested in current events and the affection which he formerly did not show toward his relatives has again returned; the restlessness and "wanderlust" have disappeared and, upon close questioning and observation, he is no longer "impelled" to lie, steal or burn things, etc.

Last Examination (October 20, 1918—18 months after operation).—No complaints. Mentality and emotional reactions normal. Both decompression openings depressed beneath flush of scalp; normal pulsation. Pupils equal and react normally. Reflexes—patellar active but equal; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi—retinal veins enlarged; no blurring of details of optic disks.

Remarks.—It would seem that the condition of this patient resulted from a brain injury at birth associated with a subdural hemorrhage, particularly over the left cerebral cortex, in the form of a supracortical layer of hemorrhage; this supracortical blood-clot became organized and, instead of forming a cyst or fibrous mass, developed a large mass of blood-vessels and thus there was formed an angiomatous tumor mass lying upon the left cerebral cortex. As a result of the diffuse subdural hemorrhage, the normal stomata of exit of the cerebrospinal fluid in the cortical veins, sinuses, etc., became blocked more or less incompletely and, as the result of this blockage, there has been an increase of the normal amount of cerebrospinal fluid within the cerebrospinal canal, and thus there was formed a condition of mild external hydrocephalus, just as results following a mild meningitis. The result of this increase of the cerebrospinal fluid has been to increase the normal intracranial pressure and thus the signs of this increased intracranial pressure were revealed in the fundi and by a measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer; the irritative presence of a supracortical angiomatous mass in the left temporo-sphenoidal area was sufficient, with the increased intracranial pressure due to the mild external hydrocephalus, to exert an irritative effect upon the cerebral cortex and thus convulsive seizures occurred. The first operation of right osteoplastic exploration sufficiently lessened the increased intracranial pressure so that convulsions no longer occurred in spite of the presence of the local irritative effect of the angiomatous tumor mass; however, as the patient became older and the cortical nerve cells developed more and more qualitatively, the still remaining intracranial pressure was of sufficient amount to pervert their normal development and particularly to impair the emotional stability, which is the most susceptible to increased intracranial pressure, whereas a distinct mental impairment does not occur unless the intracranial pressure becomes very high (this is true also of both motor and sensory impairments). Therefore, this patient could have been considered normal both physically and mentally, upon superficial examination, and yet he was definitely impaired emotionally and from this standpoint, medically, he was in reality not responsible for his conduct—no more so than if he were suffering from the toxic condition following typhoid fever, alcoholism and the other conditions which render one's emotional conduct irresponsible. If the increased intracranial pressure in this patient had not been relieved and especially the direct pressure of the overlying tumor mass, the emotional instability would undoubtedly have progressed, convulsions would probably have returned from the cortical irritation and later the mentality have become affected, so that this patient would eventually have been classed among patients mentally and emotionally deranged; if then the increased intracranial pressu
not relieved and even if it were relieved at this late date, the pr
would be most doubtful. Even at the time of operation upon this
the symptoms and signs of an incipient dementia præcox could
considered and this patient could very easily have been classed a
affected. Naturally, sufficient time has not elapsed to warr
opinion regarding the prognosis of this patient, but his

improved since the operation that the result has been most gratifying and encouraging; any patient, however, who has passed through a similar history must be considered as temporarily damaged and a period of time of at least 5 years is necessary before a competent opinion of the prognosis is possible.

C. *Mental derangement.*

CASE 128.—Old severe brain injury associated with a depressed fracture of the vault, an increased intracranial pressure and melancholy. Institutional care. Partial craniectomy; drainage of supracortical hemorrhagic cyst. Excellent improvement and recovery.

No. 043.—John. Fifty years. White. Single. Fireman. U. S.

Admitted May 10, 1913—15 years after injury. Polyclinic Hospital. Referred by Doctor George E. Brewer.

Operation May 21, 1913—11 days after admission. Partial craniectomy; drainage of cyst.

Discharged May 27, 1913—7 days after operation.

Family history negative; no insanity or nervous instability.

Personal History.—Fifteen years ago, while working as a marine engineer on a fire-boat of the New York City Fire Department, patient was struck over the top of the head by a wooden beam; "everything became very dark," but patient does not think he lost consciousness; no bleeding from nose, mouth or ears, and after lying down for several hours, he was able to continue his work. During the following 2 weeks, he had severe headaches, vomited daily, and finally became so disoriented that he roamed about the city in a dazed, semiconscious condition—unable to find his way home; patient was arrested and then transferred to Bellevue Hospital, and after remaining there one month, he was sent to Bloomingdale Asylum, White Plains, where he remained for a period of 11 months—the diagnosis being "melancholia"; while there, patient now says "I was not crazy enough to be in an asylum and yet not well enough to be at work." Finally he escaped from Bloomingdale, found light work, but was unable to hold a position longer than several weeks. Patient was again found roaming the streets, and this time he was confined in Central Islip Insane Asylum—the diagnosis being "melancholia"; he later escaped from this instituton, went out West and was finally confined in an asylum from which he escaped—only to be confined in another asylum—the diagnosis always being "melancholia"; whenever he became sufficiently lucid later to realize his surroundings, he would escape. Seven months ago while in a Massachusetts State Insane Hospital, patient was trephined in the median line of the frontal bone and an area of bone 1½ inches removed; the longitudinal sinus, however, was torn, requiring immediate packing and no further exploration or operation was possible. (It would be interesting to know just what benefit could be derived by an operation of this character and in this location, especially since the dura was not opened.) Patient finally obtained his discharge from this hospital 4 months later, and after remaining at home for 2 months in a fairly rational condition, he was referred for examination.

Examination upon admission (15 years after injury).—Temperature, 98.6°; pulse, 70; respiration, 20; blood-pressure, 142. Well-developed and nourished, but apparently much older than his real age. Morose and depressed, showing no interest in surroundings and current events. Memory for recent events was impaired and all affection for relatives lost. Complains of dull heaviness throughout the head and spells of mild vertigo. No weakness of the arms, legs or face; no sensory impairment except the loss of the sense of position of toes of both feet—more on left than on right (he could not tell whether left big toe was being pressed upward or downward). Pupils equal and react normally. Reflexes negative. Fundi—retinal veins rather full; no edematous blurring of details of optic disks, which were rather whitish and their margins slightly irregular and ill-defined; both physiological cups rather shallow from new tissue formation—that is, mild signs of a secondary optic atrophy (indicating a former increased intracranial pressure). Lumbar puncture—clear cerebrospinal fluid under slightly increased pressure (approximately 12 mm.); Wassermann test negative and cell count only 6 cells per c.mm. X-ray (Doctor A. J. Quimby)—"slight flattening of top of vault beneath which the underlying bone was rather blurred—possibly an old line of fracture at this point" (Fig. 150).

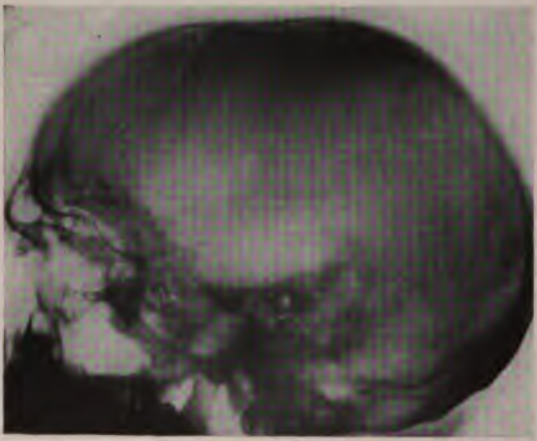

FIG. 150.—Old depressed fracture of vertex of vault in a patient becoming mentally deranged. Removal of bony depression and an underlying cortical cyst permitted an excellent recovery.

Treatment.—It was considered advisable to explore the top of the vault as suggested by the X-ray, especially since the neurological examination indicated the underlying area of the brain as the site of a possible organic lesion—the loss of the sense of position of the toes of both feet, and especially those of the left foot; the signs of a mild increase of the intracranial pressure as revealed by the ophthalmoscope and at lumbar puncture were also suggestive of an intracranial lesion, and yet not high enough to warrant a subtemporal decompression first.

Operation (11 days after admission).—Exploratory craniectomy: transverse curvilinear incision of the scalp 3 inches across the longitudinal sinus and over the vertex of the vault; a small trephine opening was made on either side of the sinus, these openings enlarged by rongeurs and finally the intervening bridge of bone over the sinus removed; in this area just to the right of the longitudinal sinus, the inner table of the vault was thickened irregularly as though resulting from a former fracture of the inner table in this area; both parietal bones at the coronal suture were thickened to a diameter of ¾ inches and were depressed at this point to a depth of ¼ of an inch. Just to the right of the longitudinal sinus, the dura bulged slightly and upon opening it, there was exposed an underlying cyst, the size of an olive, lying beneath the longitudinal sinus and betweeen the falx cerebri and the right cerebral cortex itself—indenting the latter but not to the extent of causing a cortical laceration, and adhering to the cortex (the result of a former supracortical hemorrhage in this area and most probably due to a tear of the overlying longitudinal sinus at the time of the injury). It was considered surgically unwise to attempt the removal of this cystic formation *en masse,* so that it was therefore merely punctured, and its outer wall excised, allowing 3 c.c. of straw-colored fluid to escape under tension; it was believed that the removal of the overlying bone would permit a sufficient local decompression to occur, so that, even if the cyst should refill, it would not exert its former compressive effect. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Patient made an excellent operative recovery so that he could be discharged 7 days after the operation; incision healed *per primam.*

Examination at discharge.— Temperature, 98.8°; pulse, 68; respiration, 20; blood-pressure, 148. Mental and emotional condition the same as before operation. No complaints, however, except soreness at the site of the operation. Operative site has healed perfectly and pulsates normally. It is interesting to note that the sense of position of the toes of both feet has returned and thus showing that the direct local compression of the cystic formation was responsible for this sensory impairment. Pupils equal and react normally. Reflexes negative. Fundi—retinal veins still slightly enlarged; the mild signs of secondary optic atrophy are naturally still present and will always remain, in that there has been a definite tissue formation about and in the optic disks due to the former edematous changes of the optic disks resulting from an increased intracranial pressure.

Examination (June 5, 1914—13 months after operation).—No complaints; no longer dull headaches or dizzy spells. Relatives state that he is a "changed man." Patient has been working for the past 6 months as watchman at a crossing of the Long Island Railroad. Careful physical examination negative, except for the mild signs of old secondary optic atrophy. Site of operation depressed and pulsates normally. Sense of position of toes of both feet normal. During the past 4 years, this patient has been examined at irregular intervals; his associates at work on the railroad state that he is rather "eccentric," but otherwise he does his work well and is not considered abnormal; "he is not talkative and does not make friends easily."

Last Examination (September 16, 1918—64 months after operation).—No complaints; works daily. Operative area depressed and pulsates normally. Sense of position of toes of both feet normal. Reflexes negative. Fundi—retinal veins of normal size; mild signs of old secondary optic atrophy persist.

Remarks.—The history of this patient is very instructive: at the time of the supposed trivial "bump" on the top of the head, patient undoubtedly had a fracture of the inner table of the skull with a small tear of the underlying longitudinal sinus, so that it bled subdurally and more on the right side —forming a blood-clot between the falx cerebri and the right cerebral cortex;

as the result of the organization of this blood-clot, a cystic formation occurred, causing the sensory impairment (ascertained years later). The increased intracranial pressure, due to the intracranial hemorrhage and the resulting chronic cerebral edema, was sufficient to produce the emotional instability in this patient chiefly one of depression and melancholia—to the extent that the patient was mentally unbalanced and therefore requiring institutional care. Although possibly a sufficient period of time has not elapsed to warrant an excellent prognosis, yet the improvement continues up to the present time, over 5 years since the operation, so that the outlook is very hopeful to say the least. This case emphasizes the necessity of most careful neurological examinations—particularly the ascertaining of the presence or not of an increased intracranial pressure, and then if any localizing signs can be demonstrated, the early recognition of their importance realized and the appropriate treatment advised.

(This case is also interesting from another point of view: at the time of the original injury—15 years before operation and when the patient was confined first in Bellevue Hospital, he was put upon the sick list of the Fire Department of New York City and finally a full pension granted to him; the patient, however, never received any of this pension—by devious political ways, the money reached a "ward-heeler" registered under the same name, and thus for 15 years this patient was deprived of money due him. Upon recovering his emotional stability, patient learned of this injustice, brought suit against the City of New York and in 1916—3 years after operation, the patient was given the money due him with interest.)

Case 129.—Old severe brain injury, with a depressed gunshot fracture of the vault, associated with an increased intracranial pressure and a mental derangement of the dementia præcoid type. Right subtemporal decompression and removal of the depressed area of vault; supracortical angioma. Only a temporary improvement.

No. 620.—John. Twenty years. White. Single. School. U. S.

Admitted May 26, 1916—4 years after injury. Alexian Brothers Hospital, Elizabeth, N. J. Referred by Doctor Otto Wagner.

Operation June 29, 1916—33 days after admission. Right subtemporal decompression; removal of depressed area of vault.

Discharged July 16, 1916—17 days after operation.

Family history negative.

Personal History.—Always well and strong until 4 years ago, when the patient was accidentally shot twice through the head—one bullet entering the right temporal region and passing obliquely upward and out through the vertex in the midline, while the other bullet entered beneath the left eye and lodged just anterior to the sella turcica. Unconscious for several days; seven general convulsions occurred and then he made a slow and gradual recovery; never the same, however, as before the injury. Patient complained of dull headache during the following 2 years, and then a noticeable change in the patient appeared; he no longer complained of headaches, but he became morose and gloomy, showed no interest in anything, no longer affectionate—so that the diagnosis of incipient dementia præcox was considered. During the past 2 years, this condition has remained practically the same until one month ago, when the patient again complained of headaches and has vomited several times; no convulsions, however, since the injury itself.

Examination in consultation with Doctor Wagner.—Temperature, 98.8°; pulse, 70; respiration, 18; blood-pressure, 130. Well-developed and nourished. Apparently rational when questioned, but he would lapse quickly into moods of depression—no longer interested in anything. Complains of continuous throbbing headache, chiefly over the frontal region. Irregular area of bone over midline of vault just posterior to vertex—the scar of exit of one bullet; small scar just below the left orbit. No motor nor sensory impairment; no impairment of joint sense nor of sense of position of extremities. No astereognosis nor apraxia. No signs of subtentorial lesion—nystagmus, ataxia nor Romberg sign, etc. Pupils equal and react normally. Reflexes: patellar exaggerated—left more than right; left exhaustible ankle clonus and left Babinski; abdominal reflexes present and apparently equal. Fundi —retinal veins dilated; nasal halves of both optic disks obscured by edema. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 18 mm.); Wassermann test negative and cell count was 8 cells per c.mm. X-ray report—"Posterior to the scar at the vertex of vault is an irregular area of bone due to an old fracture and beneath it is an irregular light area, almost 2 inches in diameter; a bullet can be seen just anterior to the sella turcica and its pathway is indicated by fragments of lead—this being the injury of the second bullet at the time of the original cerebral injury."

Treatment.—Although the mental and emotional condition of the patient was such that the diagnosis of dementia præcox was very possible, still on account of the signs of an increased intracranial pressure, the definite history of cranial injury and the X-ray picture showing a lesion beneath the fracture of exit at the vertex of the vault, it was decided to perform first a right subtemporal decompression and then to explore at the site of the fracture on the vertex.

Operations (4 years after injury).—First. Right subtemporal decompression: usual vertical incision, bone removed, and no complications. Dura very much thickened, vascular and tense—numerous large sinuses throughout dura, giving it a corrugated appearance; upon incising dura, clear cerebrospinal fluid spurted to a height of 2 inches, revealing a very "wet," edematous cortex with many newly formed vessels lying upon it and in a cystic formation of the arachnoid (being very similar to the pathological picture resulting from a supracortical hemorrhage and observed most frequently in conditions of cerebral spastic paralysis due to a supracortical hemorrhage at the time of birth). A large amount of cerebrospinal fluid escaped so that the cortex protruded less tensely and pulsated at the end of the operation. Usual closure with one drain of rubber tissue inserted. Temporary sterile gauze dressing applied.

Second Operation.—A transverse curvilinear scalp incision now made just posterior to the vertex and extending across the median line; a small trephine opening made on either side of the longitudinal sinus, these openings enlarged and then the bone overlying the sinus itself was removed. A most unusual dural picture presented itself; large dural sinuses, the size of lead pencils, extended to the right of the longitudinal sinus, forming a tumor mass similar to a "bunch of fish-worms" (the cause for the irregular light area shown in the X-ray pictures). The intradural pressure in this area did not seem to be abnormally increased (due to the preceding subtemporal decompression), and, therefore, the dura was not opened; the danger also of attempting such a procedure on account of the angiomatous mass would have been very great indeed. Usual closure with 2 drains of rubber tissue inserted. Duration, 90 minutes.

Post-operative Notes.—Patient made an excellent operative recovery so that he was discharged from the hospital 17 days after operation; operative incisions healed *per primam.* During the following 12 months, patient was improved in that he no longer complained of headaches and was of a more cheerful disposition; he was, however, unable to concentrate mentally. Twelve months after operation, patient entered again into a state of depression, no longer interested in his surroundings, and was eventually recommended for institutional care—the present diagnosis being dementia præcox. X-ray picture "discloses the bullet and its pathway of entrance; also three silver clips within the oval decompression area" (Fig. 151).

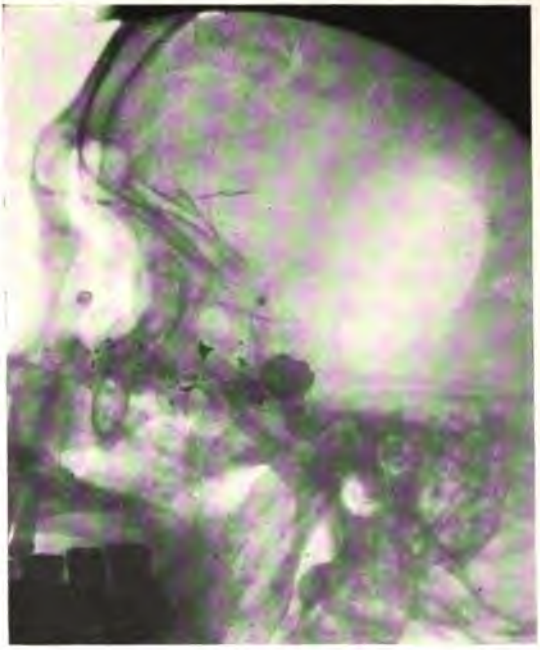

Fig. 151.—Revolver bullet and its pathway beneath left orbit in a patient shot twice in the head; mental derangement. Temporary improvement only, following a right subtemporal decompression, which can also be seen in the röntgenogram.

Remarks.—Whether this patient, if operated upon early, could have been spared this emotional instability cannot be assured; it does seem reasonable, however, that if a lessening of this increased intracranial pressure could have been secured and especially if the supracortical hemorrhage resulting from the bullet injury in its intracranial passage could have been drained by a subtemporal decompression immediately after the injury, that the patient would
have had a much greater chance of ultimate recovery of his f
mality; merely because this patient now has the symptoms a
dementia præcox, this does not mean that his condition is o
and essentially a true dementia præcox—with its most hopele
This case merely emphasizes the necessity of proper surgic
following a cranial injury having marked signs of increase
pressure; practically all gunshot injuries penetrating the ir

produce a subdural hemorrhage and this hemorrhage should always be drained; this is especially true when it is associated with an increased intracranial pressure, due not only to the hemorrhage alone but to the resulting traumatic cerebral edema.

The presence of the bullet intracranially and in the neighborhood of the sella turcica is not exhibiting any clinical signs and naturally no attempt should be made to remove it. Even if it should be within the brain substance, any operation performed to remove it would merely increase the damage to the patient, and if no signs of its presence existed before the operation, these signs would most probably appear after the operation and as a result of the operative damage to the cerebral tissues.

Case 130.—Old severe brain injury associated with a depressed fracture of the vault and with signs of an increased intracranial pressure; emotional and mental impairment of the traumatic dementia type. Left subtemporal decompression. Improvement.

No. 1045.—Albert. Thirty-two years. White. Married. Pattern-maker. United States.

Admitted December 3, 1918—27 years after injury. Audubon Hospital. Referred by Doctor E. L. Kellogg.

Operation December 18, 1918—15 days after admission. Left subtemporal decompression.

Discharged January 5, 1919—18 days after operation.

Family history negative.

Personal History.—Patient was considered a normal child until cranial injury. When 5 years of age (27 years ago), patient was kicked over the left frontal region by a horse; immediate loss of consciousness; no bleeding from nose, mouth or ears; patient was kept in bed for one week, when the irregular depressed bone of 1½ inches in diameter was removed; an excellent recovery apparently, and the patient was considered normal until 6 years ago (at the age of 26 years), when he began to complain of headache, inability to "concentrate his mind," early fatigue and an increasing depression and melancholia; he refused to work for fear of it "hurting" him. This condition of mental and emotional deterioration increased, so that during the past 2 years he will not associate with anyone, sits by himself sullenly, displays no affection toward friends or relatives, says he is worried and has even suggested suicide as a means of relief; that is, he displays many of the symptoms and signs of dementia præcox, and also of traumatic dementia, in that his memory has become impaired, says childish and "foolish" things and must now be accompanied upon the street for fear he will become lost. He has always complained of dull headache and a heavy feeling in his head.

Examination upon admission (27 years after injury).—Temperature, 98.6°; pulse, 70; respiration, 18; blood-pressure, 132. Fairly well-developed and nourished. Very much depressed and melancholic; answers questions after hesitation and with much difficulty; definite impairment of memory and says "I fear I shall not last long." Over the left frontal bone is an irregular scar and a definite depression at the site of the former trephine opening. No paralyses or sensory impairments. Hearing negative; otoscopic examination negative. Pupils equal and react normally. Reflexes—patellar active, right possibly greater than left; no ankle clonus but suggestive right Babinski; abdominal reflexes present and equal. Fundi—retinal veins enlarged; nasal margins of both optic disks blurred by edema; both physiological cups shallow from new tissue formation and both optic disks rather pale from an increased formation of new tissue—a very mild degree of secondary optic atrophy, right possibly more advanced than left; visual acuity, however, was 15/20 in each eye. Lumbar puncture—clear cerebrospinal fluid under increased pressure (14 mm.); Wassermann test negative and cell count was 6 cells per c.mm. Urine examination negative. X-ray (Doctor A. J. Quimby)—"an irregular bony defect of left frontal area; no linear fracture visible" (Fig. 152).

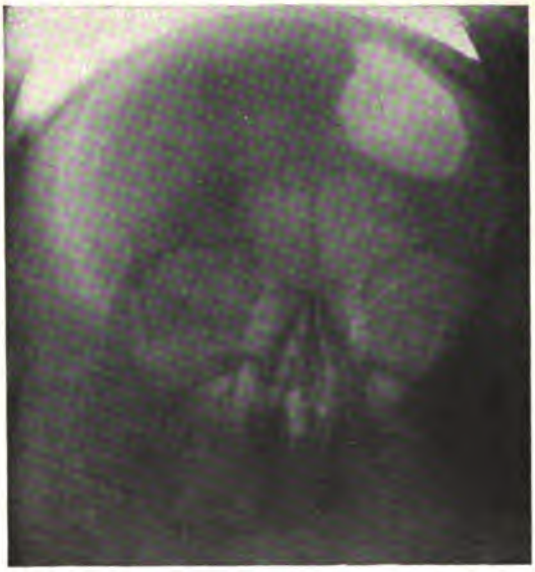

FIG. 152.—Irregular bony defect of left frontal area in a patient having an old depressed fracture and subsequent mental derangement. Improvement following an operative lowering of the increased intracranial pressure.

Treatment.—On account of the definite signs of an increased intracranial pressure, it was considered advisable to perform a left subtemporal decompression in the hope that the condition of this patient could be improved; the long period since the injury and the marked emotional and mental impairment are naturally factors which make the ultimate prognosis very doubtful; the absence of convulsions, however, is encouraging.

Operation (27 years after injury).—Left subtemporal decompression: usual incision, bone removed, and no complications. Dura whitish and under moderate tension; upon incising it, clear cerebrospinal fluid spurted to a height of 1 inch and upon enlarging the dural opening, a very "wet" edematous cortex tended to protrude but did not rupture; much cerebrospinal fluid escaped, permitting the cortex to recede before the end of the operation. In the arachnoid and about the cortical vessels in the sulci was a grayish fibrous induration—the residue of a former subarachnoid hemorrhage. No laceration or cystic formation visible. Usual closure with 2 drains of tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Uneventful operative recovery; incision *per primam* and the patient was discharged on the eighteen after operation.

Examination (February 20, 1919—2 months after operation).- ing to the father and the patient himself, he has made a marked ment in that he has been working the past week, is showing an increasing interest in his work, and says "I am going to do well." Father states that patient is not so depressed, is becoming interested in his two children and in every way he is more like his former self. Decompression area is flush with the surrounding scalp. Reflexes active but otherwise negative—there being no left Babinski elicitable. Fundi—retinal veins rather full; very indistinct edematous blur of lower nasal quadrant of both optic disks—much less than before the operation; the new tissue formation of the mild secondary optic atrophy naturally still persists.

Last Examination (December 8, 1919—12 months after operation).—Father writes that patient has been working daily and no "troublesome complaints; in many ways, he is a changed man."

Remarks.—It would be most surprising and unusual if this patient would make an excellent recovery both emotionally and mentally, and yet I feel he has an excellent chance of being markedly improved; it may be of only temporary duration, but even then the operation would be justified if his condition were only improved for a period of a year or more, and the condition retarded and delayed. In the absence of the cranial injury, and particularly if there were no signs of an increased intracranial pressure, then the diagnosis would undoubtedly be that of dementia præcox with its hopeless prognosis; the ascertaining, however, of the signs of a definite increase of the intracranial pressure immediately withdraws this patient from that large hopeless group of patients having dementia præcox and makes it possible for him to be improved by means of a mechanical lessening of this increased intracranial pressure; his condition may not be ultimately improved and yet the operation gives him a definite chance. A report of this patient will be made later after a period of several years has elapsed.

Merely removing a small area of depressed bone of the vault without opening the dura widely in these patients having an increased intracranial pressure associated with the depressed fracture of the vault, is very incomplete surgical treatment, and it may be considered of only temporary benefit to the patient. The ideal time for the appropriate treatment of these patients having an increased intracranial pressure is immediately after the cranial injury, when the shock has subsided; then, the increased intracranial pressure can be first relieved by a subtemporal decompression and drainage, and then the depressed area of the vault can be elevated or removed. This method of treatment will prevent or, at least, tend to prevent such unfortunate results as in this patient.

PART III

ACUTE AND CHRONIC BRAIN INJURIES IN NEWBORN BABIES AND CHILDREN

CHAPTER XIII

General Considerations.—In newborn babies, acute brain injuries are the result of trauma at the time of parturition, which may be either a difficult prolonged one with and without the use of instruments or even a so-called "precipitate" birth, in that the delivery is an unusually rapid one complicated by a rupture of the thin-walled cortical veins; damage to the delicate intracranial structures may also occur in an apparently normal labor; these observations and diagnoses have been frequently confirmed by autopsies.

It has long been recognized that prolonged difficult labor, and especially if instruments for delivery are necessary, is of risk to the immediate recovery of life of the child; this danger to life itself has been comparatively slight, but if the death of the child did occur, then it was realized (and occasionally confirmed at autopsy) that the intracranial contents had been so badly damaged that even if the baby had recovered, yet it could not have been a normal child mentally and physically, and therefore it was merely considered an unavoidable and unfortunate result of a difficult labor, the object being to secure a living mother damaged as little as possible and then a living child, if possible. If the child was successfully resuscitated immediately after birth, so that it was considered normal and not damaged intracranially, the prognosis as to future normality was naturally excellent—and in fact, this is the usual result. Even if the child was drowsy and stuporous for a period of ten days and longer, when it did not cry as newborn babies ordinarily do, or if it was of the excitable restless type and crying almost continuously and whether slight convulsive twitchings of any part of the body were present or not—this condition during an indefinite period of days following delivery was usually a temporary one only, so that it was not considered as being permanently harmful to the future of the child; in other words, the child "would grow out of it." And in the majority of babies with this immediate post-traumatic history, the condition does gradually disappear and fortunately no ill-effects are later to be observed due to the entire absorption of the intracranial hemorrhage and cerebral edema. There is a small percentage of babies, however, in which this happy result does not occur; either they remain in a comatose condition with and without convulsive seizures for several days and then die—and at autopsy an extensive subdural and usually a supracortical layer of hemorrhage is revealed associated with a very "wet" edematous condition of the brain, or they apparently become normal within several days, and are considered so until the sixth, seventh, eighth or ninth month later, when it is realized that the child is not developing as a normal child should—is not holding up its head, does not attempt to grasp and to hold things, notices little if anything, etc., and as it becomes older this retardation and impairment, both physically and mentally, becomes more and more marked; even at this late date of months and especially without careful examinations, the parents may be told that nothing is really "wrong" with the child, "merely retarded," and "it will grow out of it." These children, however, rarely do become normal when the condition is the result of a large intracranial lesion at the time of birth—usually a supracortical layer of hemorrhage with little or no primary damage to the brain itself, and in the babies which cannot "take care of" this large amount of hemorrhage and cerebral edema by the natural means of absorption, then the effects of this intracranial condition associated with a definite increase of the intracranial pressure are later shown in a general retardation of the development of the child, both mentally and physically. Unless this increased intracranial pressure of hemorrhage and excess cerebrospinal fluid is relieved early—if not immediately after birth then within several days, or if the condition is permitted to continue until the latter months of the first year and even later, then its lowering by means of a subtemporal decompression, and if necessary a bilateral decompression is essential in order to permit a marked improvement to occur, although the longer this increased intracranial pressure is allowed to continue, either through ignorance, carelessness or mistaken diagnosis and judgment, just so much more permanent is the cerebral impairment in its mental and physical results. The differential diagnosis at this late date is between that of lack of development of the cerebral cortex or its pyramidal tract fibres (the so-called Little's Disease), a meningitic and meningo-encephalitic destructive process associated or not with embolic or thrombotic complications, hereditary lues (less than 2 per cent.) and then the condition of intracranial hemorrhage at the time of birth and of such large amount, that the natural means of absorption have not sufficed to permit the normal lowering of the increased intracranial pressure of the hemorrhage and the chronic cerebral edema, resulting from a partial blockage of the stomata of exit of the cerebrospinal fluid in the cortical veins, sinuses, etc., by the organization of this layer of supracortical hemorrhage; and thus in reality, producing a mild external hydrocephalus similar, but in milder form, to the condition of hydrocephalus resulting from an extensive meningitic process, which does not block the ventricles and therefore producing the more common type of external hydrocephalus. These chronic brain injuries occurring in children who become impaired both mentally and physically and particularly of the type of cerebral spastic paralysis, will be discussed later in detail under the heading of chronic brain injuries in children.

In the acute brain injuries with and without a fracture of the skull occurring in children under 12 years of age, the immediate effects of intracranial lesions can be withstood much more successfully than in adults—the initial shock is less, the reaction is much stronger and prolonged, and they can recover from intracranial trauma as far as immediate life is c cerned much more easily and with fewer immediate complications t is possible in adults; but the remote effects, however, of serious intracra lesions in children, and especially associated with a prolonged increase of intracranial pressure, are more permanent in these patients later in life. they form a very influential factor in the future development of the ch both mentally and physically. It is this remote factor and re intracranial injuries in childhood which have been rather neglected and overlooked.

A. Acute Brain Injuries in Newborn Babies.—It is not uncommon for acute brain injuries to occur in newborn babies during parturition—usually a difficult labor with and without the use of instruments, but it is rare for these intracranial lesions to be associated with a fracture of the skull; occasionally depressed fractures of the vault and of the so-called "ping-pong" type result, but it is most unusual for a frank linear fracture of the flexible newly-formed bone to be demonstrated—by röntgenograms, operation or at autopsy. If there is present in these newborn babies any abnormality of the bones of the skull, then it is almost invariably a diastasis and separation of the suture lines with and without their overlapping, one over the other; the suture line most frequently involved is the median one between the two parietal bones and overlying the longitudinal sinus, which may thus be torn, permitting an intracranial hemorrhage of varying size to form over the cortex of one or both hemispheres of the brain—and this is a very common type of intracranial hemorrhage occurring in newborn babies as a result of the change of continuity of the bones of the vault. This separation of the suture lines and the overlapping of the adjacent bones rarely persist after birth longer than hours or days at the most and they may be present only during the active second and third stages of labor, and then the bones resume their normal relation and position—but after the damage to the sinus has resulted. This is the reason why careful bimanual examination of the heads of these children and the still later röntgenograms in various planes only infrequently demonstrate the presence of the overlapping of the lines of suture. The frontal bone in its posterior relation to the parietal bones to form the coronal suture and the occipital bone in its anterior relation to the parietal bones to form the lambdoidal sutures are the next most common sites for the overlapping of their respective suture bones, and yet intracranial lesions only occasionally follow since there are here no underlying sinuses.

If the longitudinal sinus is not torn and it is possibly one of the most frequent causes of the condition, then the next most usual source of the supracortical hemorrhage is a rupture of the delicate supracortical veins of either or both cerebral hemispheres, as a result of a severe venous stasis and congestion occurring during a prolonged difficult labor; the hemorrhage may be only a local one—the size of a ten-cent piece or a silver quarter, and yet the associated cerebral edema following the cerebral trauma is always present, and may in many patients be the more serious factor. It is thus seen that the intracranial hemorrhage in these newborn babies rarely occurs in the cerebral cortex and in the brain itself, and therefore causing a primary destruction of brain tissue (and no regeneration), but the hemorrhage is almost always subdural but supracortical—lying upon the surface of the brain—and its damage to the underlying cerebral cortex is one of pressure, due both to the hemorrhage itself and to the resulting cerebral edema; that is, if this supracortical hemorrhage and excess cerebrospinal fluid can be successfully drained and thereby the increased intracranial pressure be permanently lowered, then these babies will have an excellent opportunity to recover—not only the immediate recovery of life but that of future normality—and *now* is the ideal time for the appropriate treatment of these patients; later in life the impaired condition can be improved, but rarely is a perfectly normal child then possible.

No doubt, there are many cases of latent intracranial hemorrhage at birth, where there are no marked clinical signs of the presence of the lesion and where the natural means of absorption are sufficient to "take care of" the mild increase of the intracranial pressure—and a normal child is possible. And on the contrary, later impairments occurring in certain children in adolescence, such as mild mental retardation, emotional instability and even epilepsy itself, may be due to a mild intracranial hemorrhage at the time of birth; its mild clinical signs not recognized or being overlooked, and the later appearance of signs indicative of a former intracranial lesion with resulting adhesions, etc., and—it is then usually too late to obtain a very satisfactory result by any treatment now known—the child may approximate normality but scarcely ever attain it. The treatment should be preventative whenever possible; if not possible, then at least corrective.

Although the labor itself in these babies developing an intracranial hemorrhage at the time of birth need not be a prolonged difficult one associated with the use of forceps, yet it very frequently is; also, the condition itself occurs most often in first babies of full term. The condition occasionally results even from a so-called normal delivery, although a difficult labor with and without the use of instruments is the usual history obtained. Any newborn baby which does not behave normally within the first two or three days after birth, in that it is unusually drowsy and even stuporous. and especially in the presence of convulsive twitchings of any part of the body—that baby should be carefully examined for definite signs of an intracranial hemorrhage; a lumbar puncture is of the greatest diagnostic importance; if free blood is found in the cerebrospinal fluid, not only is the diagnosis confirmed but an excellent means of drainage is thus afforded unless the pressure is high—over 15 mm. as registered by the spinal mercurial manometer and associated with tense fontanelles and positive ophthalmoscopic findings. Daily repeated lumbar punctures with removal of 10–12 c.c. of bloody cerebrospinal fluid may be performed upon a number of consecutive days until the pressure of the cerebrospinal fluid does not exceed 10 mm. and in these patients an excellent result is frequently obtained. In those babies, however, in whom the increased pressure of the cerebrospinal fluid reaches a height of 15 mm. and even higher, and especially when associated with tense fontanelles and positive ophthalmoscopic findings of increased intracranial pressure, then a modified subtemporal decompression and drainage is most advisable in order to obtain not only a living child but of the greatest importance—a normal child later.

Through the usual vertical incision of the subtemporal decompression with retraction of the split temporal muscle, a small area of bone—not larger than a silver quarter, may be rongeured away and then the dura carefully opened; the supracortical hemorrhage and excess cerebrospinal fluid can thus be easily drained; a small drain of rubber tissue is inserted beneath the temporo-sphenoidal lobe into the middle fossa of the base, in order to continue the drainage for one or two days—the usual length of time sufficient to lower the intracranial pressure permanently. No anesthesia or primary anesthesia alone is required, and only at the very beginning of the precedure—in order to insure the operative asepsis. In some selected patients whose pressure of the cerebrospinal fluid does not exceed 18 mm., then through a similar but smaller vertical incision of 2 cm. over the squamo-parietal suture line, an opening through the membranous suture line may very easily and quickly be made with a scalpel and tissue forceps, the dura carefully incised and a drain of rubber tissue inserted. The objections to this method are that the drainage opening is higher and therefore nearer the motor area of the cerebral cortex with a consequent greater danger of operative damage to the underlying highly developed and more important portions of the brain, and also that the drainage itself is not so satisfactory as when the drain can be inserted into the middle fossa at the base. This method of drainage through the suture line is no longer used in our clinic. Naturally, the right side of the head in these patients is operated upon, if the parents are both right-handed and if there are no localizing signs indicative of a greater lesion upon the opposite side of the head.

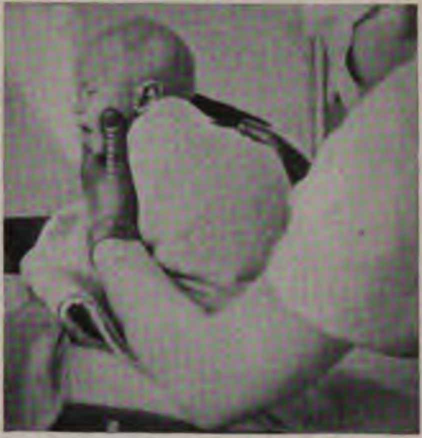

FIG. 153.—Large depressed fracture of left parietal area following an instrumental delivery in a baby of five weeks of age, showing a definite weakness of the right side of the body. Complete recovery following the removal of the depressed bone.

CASE 131.—Recent depressed fracture of the left parietal area of the vault of the skull in a newborn child; localizing symptoms and signs. Removal of depressed area of vault. Excellent recovery.

No. 957.—Olga. Five weeks. White. United States.

Admitted March 4, 1918, 5 weeks after injury. Polyclinic Hospital. Referred by Doctor J. V. D. Young.

Operation March 5, 1918. Removal of depressed bone.

Discharged March 10, 1918, 5 days after operation.

Family history negative.

Personal History.—First baby, full term, instrumental delivery. Immediately after birth, it was noted that the left parietal area of the vault was depressed two inches in diameter and one-half inch in depth. No bleeding from nose, mouth or ears; no mastoid ecchymoses. Child was rather drowsy and stuporous, but otherwise apparently normal; no clinical signs of localization referable to the left cerebral cortex; no convulsive seizures. Fontanelles rather tense and bulge slightly.

First Examination (February 26, 1918—4 weeks after birth).—Temperature, 98.8°; pulse, 104; respiration, 28. Well-developed child. Rather sleepy and drowsy—does not cry upon being disturbed. In left parietal area of the vault is a depression of almost one-half inch in depth and of two inches in diameter (Fig. 153); no pulsation palpable. Fontanelles rather tense but pulsation normal. Definite weakness of right arm, slight weakness of right leg, and a possible flaccidity and flatness of the lower portion of right side of face (the cortical type of facial paralysis). Sensation—no impairment could be elicited. Pupils equal and react normally. Reflexes: patellar right possibly more active than left; no ankle clonus but double Babinski; abdominal reflexes cannot be elicited. Fundi: retinal veins full and tortuous—left possibly more than right; definite edematous blurring and haziness of nasal margins of both optic disks—left greater than right. Lumbar puncture—clear cerebrospinal fluid under slightly increased pressure (10 mm.). X-ray (Doctor G. W. Welton)—"depressed area of left parietal bone of one and one-half inches in diameter; no comminution of fracture observed" (Fig. 154).

Treatment.—In the presence of a depressed area of the vault producing the definite localizing signs of weakness of the opposite side of the body and the fear that convulsive seizures might later occur as a result of the irritation of the underlying cerebral cortex, it was considered advisable and imperative to elevate and, if necessary, to remove the depressed area of bone (which should have been performed within several days after birth).

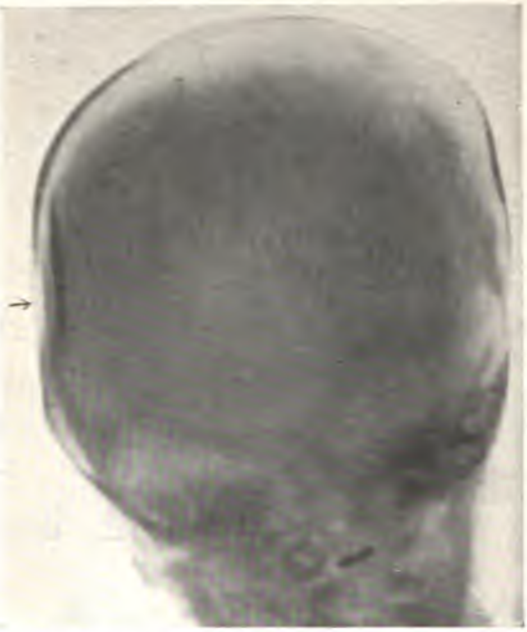

FIG. 154.—Depressed fracture of left parieto-squamous area in a newborn baby producing a right hemiparesis. Complete recovery following an operative removal of the depressed bone.

Operation (March 5, 1918—5 weeks after injury).—Removal of depressed area of vault, primary anesthesia alone being administered: vertical incision of two inches over depressed area of bone of the left parietal region; scalp retracted and an effort made to elevate the depressed bone by means of a small periosteal elevator being inserted through the line of fracture at the periphery of the depression; this attempt was not successful and the depressed bone was now rongeured away to a diameter of
one-half inches. The intact underlying dura which had been d
so that it was concave, now welled upward and assumed its normal
ity. Dura slightly tense but pulsated normally, and therefore it
opened; it was of normal appearance. Usual closure with two
rubber tissue inserted down to dura. Fine interrupted silk sutures
to approximate the scalp in one layer. Duration, 25 minutes. Post-

notes: uneventful recovery. Within 48 hours, the definite weakness of the right side of body had become less marked and at the end of 4 days no impairment could be ascertained. Child became less drowsy and stuporous and presented no abnormality.

Examination at discharge (5 days after operation).—Temperature, 98.6°; pulse, 98; respiration, 28. Operative incision healed *per primam;* it is flush with the surrounding scalp and pulsates normally. No weakness of the right side of the body can be elicited. Pupils equal and react normally. Reflexes: patellar—active, right possibly greater than left; no ankle clonus but double Babinski; abdominal reflexes absent. Fundi: retinal veins slightly enlarged; very faint obscuration of nasal margins of both optic disks.

Examination (October 20, 1918—7 months after operation).—No complaints; "a normal baby in every way." Operative area slightly depressed; normal pulsation; some new bone formation at periphery of the bony opening. Pupils equal and react normally. Reflexes: patellar—active but equal; no ankle clonus but indefinite double Babinski; abdominal reflexes—both depressed but equal. Fundi negative.

Last Examination (March 6, 1919—12 months after operation).—No complaints. Operative area slightly depressed and of smaller diameter due to new bone formation at periphery; only slight pulsation palpable. Reflexes negative. Fundi negative.

Remarks.—This is the type of patient having a depressed fracture of the vault at the time of birth which is frequently not elevated or removed, in the belief that the normal convexity of the vault will be later approximated by the growth of the brain gradually forcing the depressed area of bone outward; that is, these depressed fractures of the vault occurring in newborn babies "will take care of" themselves. No doubt a large number of these patients make excellent recoveries, both as to immediate impairment and to future complications, but it is most unwise to permit these patients to run the great risk of serious complications and especially of convulsive seizures, before an attempt is made to elevate or remove the depressed area of bone; the risk of the operation is practically nil, especially when the dura is not opened and it never is, unless the intracranial pressure is high, and then it is better surgical judgment to perform a subtemporal decompression first and then remove the depressed area of the vault. Unless the depressed area of bone becomes elevated by itself within a week after birth, and especially if clinical signs of its presence are indicated by an increased intracranial pressure and the localizing signs of paralysis, convulsive twitchings, etc., then the local operation of elevation or removal of the depressed area should be performed without delay; to wait until the underlying brain elevates the depressed area of the vault by its normal pulsation—the brain being allowed to act as a "crow-bar," as I recently heard it expressed by a children's specialist—is hardly a duty to be imposed upon the brain and one which may result disastrously to its future normality.

CASE 132.—Acute severe brain injury in a newborn baby associated with a supracortical hemorrhage and convulsive twitchings; an increased intracranial pressure. Left subtemporal decompression and drainage. Excellent recovery.

No. 731.—William. Twelve hours. White. U. S.

Admitted November 23, 1916—12 hours after birth and injury. Polyclinic Hospital. Referred by Doctor M. A. Swiney, Bayonne, N. J.

Operation November 24, 1916—24 hours after admission and 36 hours after birth. Left subtemporal decompression and drainage.

Discharged December 2, 1916—8 days after operation.

Family history negative.

Personal history negative.

Present Illness.—First child, full term, head presentation, very difficult labor of 48 hours, requiring instruments. Weight—ten and a half pounds. Very difficult to resuscitate and he remained in a drowsy stuporous condition for 2 hours, when convulsive twitchings of the right facial muscles and of the fingers of the right hand occurred and persisted for five to seven minutes each time; six hours after birth, a convulsive seizure of the right side of the body began in the right arm and lasted for four minutes; twenty minutes later, a second convulsive seizure of the right side of the body appeared and it became a general convulsion, lasting for 2 minutes. On the way to the hospital, another general convulsion began in the right arm, then the right leg, and finally both sides of the body convulsed tonically and then clonically for a period of almost 4 minutes.

Examination upon admission (12 hours after birth and the injury).—Temperature, 104°; pulse, 120; respiration, 34. Conscious but rather drowsy. Over the posterior portion of the right frontal bone was a small hematoma—the size of an English walnut; rather tense. No bleeding from nose, mouth or ears. Left orbit swollen and ecchymosed; left subconjunctival hemorrhage. An apparent left facial paralysis of the peripheral type (left forehead muscles being involved). No paralyses of the extremities of either side of the body. Otoscopic examination negative. Fontanelles moderately tense and slightly bulging. Pupils equal and react to light normally. (During the examination the muscles about the right eye and right side of mouth twitched spasmodically and also the fingers of the right hand, but no convulsive seizure occurred.) Reflexes—patellar active but equal; no ankle clonus but double Babinski; abdominal reflexes could not be elicited. Fundi—nasal halves and temporal margins of both optic disks obscured by edema; retinal veins enlarged. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 12 mm.); cell count was 5 cells per c.mm.—no red blood corpuscles being observed. X-ray (Doctor W. H. Stewart)—"no fracture revealed."

Treatment.—In the absence of blood in the cerebrospinal fluid at lumbar puncture and since the intracranial pressure was not registered as being above 15 mm. and there having been a cessation of the convulsive seizures during the past 2 hours, it was decided to treat the child expectant the hope that the intracranial condition could be "taken care of" natural means of absorption; the usual routine treatment was used sisting of an ice-bag to the head, external warmth and absolute quiet condition of the child remained practically the same during the n hours, and it was thought that an excellent recovery both of life and mality would be possible; 18 hours after admission and 30 hours aft the third general convulsion occurred, beginning in the right arm, then the right leg, and finally both sides of the body; a fourth similar convulsive seizure occurred two hours later after which it was observed that the right arm and right leg were more lax and limp than the left arm and left leg; a lumbar puncture at this time removed clear cerebrospinal fluid under an increased pressure (approximately 14 mm.) and the cell count contained numerous red blood corpuscles; the ophthalmoscopic examination persisted in revealing an edematous obscuration of the nasal halves and temporal margins of both optic disks, but not a measurable swelling to the degree of papilledema (1 or 2 diopters). On account of these signs of an increasing intracranial pressure with the definite localizing signs pointing to the left cerebral hemisphere, it was now considered advisable to perform a left subtemporal decompression and drainage.

Operation (36 hours after birth and injury and 24 hours after admission).—Left subtemporal decompression and drainage (no anesthesia being required except codeine, grs. 1/12, hypodermically): usual vertical incision, bone removed, and no complications. Dura very tense, bulging and bluish; upon incising it, bloody cerebrospinal fluid spurted to a height of 2 inches, and upon enlarging the incision a thin layer of supracortical hemorrhagic clot welled through the dural opening, revealing a very "wet," edematous cortex which tended to protrude but did not rupture; one small supracortical vein lying in a sulcus ruptured owing to the high cerebral tension, but its bleeding was quickly stopped by the application of a small piece of temporal muscle (thus causing the rapid coagulation and blockage of the bleeding point). Much bloody cerebrospinal fluid escaped, permitting the cortex to recede at the end of the operation and to pulsate almost normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 16 minutes.

Post-operative Notes.—Uneventful recovery; profuse discharge of cerebrospinal fluid ceased 18 hours after the operation so that the drains were then removed. No convulsive seizures or localized twitchings occurred after the operation and the definite weakness of the right arm and right leg could not be elicited within 12 hours after the operation; the edema of both optic disks subsided, so that on the second day after operation only an indistinct blurring of the lower nasal margins of the optic disks could be observed. The child rapidly became more lively and on the third day after operation, he cried for the first time.

Examination at discharge (8 days after operation).—Temperature, 99°; pulse, 116; respiration, 32. Apparently normal in every way. Hematoma over the left frontal bone has disappeared. Weakness of the left side of face has lessened so that it can only be elicited while the child is crying. Decompression area bulges slightly beyond the flush of scalp; normal pulsation. No weakness of right arm or of right leg can be obtained. Pupils equal and react to light normally. Reflexes—patellar active but equal; no ankle clonus but double Babinski persists (practically normal for young babies, as it appears in a large number of them); abdominal reflexes—depressed but equal. Fundi—retinal veins enlarged; lower nasal margins of both optic disks slightly blurred by edema.

Examination (February 20, 1918—15 months after operation).—Child did not hold its head up until 8 months after birth and did not sit up until the thirteenth month; it is now crawling about and attempting to stand and to walk, but it is unable to do so. No convulsive seizures have occurred since the operation. Child is possibly not so alert mentally as it normally should be. Decompression area flush with surrounding scalp; normal pulsation. No weakness of the extremities or sensory impairments ascertained. Reflexes: active but otherwise negative; a tendency possibly to a right Babinski. Fundi negative.

Last Examination (June 6, 1919—31 months after birth and operation).—Child has progressed rapidly during the past year; he is able to walk, but has some difficulty in balancing himself. (Fig. 154*a*.) He is able to say a number of words and small sentences. No convulsive seizures have occurred; mentally not so alert and interested in his toys and surroundings as is possibly normal. Decompression area slighty depressed beneath the flush of scalp; some new bone formation at the periphery; normal pulsation. Reflexes active but otherwise negative. Fundi negative.

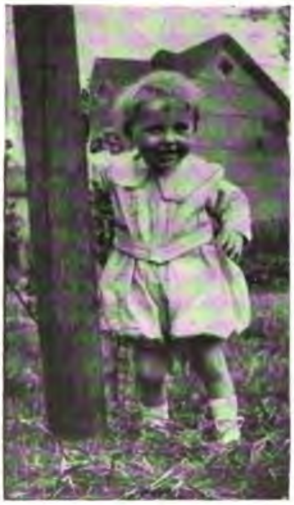
FIG. 154*a*.—Patient 2½ years after left subtemporal decompression.

Remarks.—In the hope that this baby would be able to "take care of" the intracranial lesion of cerebral edema and intracranial hemorrhage (and many newborn babies are able to absorb the milder conditions), the operation of decompression and drainage was postponed 24 hours in this patient, and it was only upon the development of the signs of an increasing intracranial pressure with the return of the convulsive seizures that made the operation an urgent one—not only in order to obtain a recovery as far as life was concerned, but also to obtain a normal child if possible. The lesion as disclosed at the operation was one from which a normal child is possible, and the steady marked improvement in this baby would make us hopeful that a normal child will be possible; sufficient time, however, has not elapsed to permit an accurate statement to be made, and it will require at least 15 years and even longer for the later condition of this patient to be ascertained; the end result may not be a normal child and may at most only approximate one, and yet I believe that the operative indication remains the same, and if children of this character can be spared the frightful condition of spastic paralysis in its various forms and mental impairment in marked degree, then there can be no question as to the advisability of the operative procedure. These patients should impress the profession with the need and the importance of most careful obstetrics and that any newborn baby, with or without a difficult labor, which does not appear to be as normal as it should be immediately after birth and particularly in the presence of convulsive seizures, then these babies should be most carefully examined, both with the ophthalmoscope and by means of repeated lumbar punctures, in order to ascertain the presence or not of an increased intracranial pressure—whether due to an intracranial hemorrhage or severe cerebral edema, and if it is thought that a normal child is not possible without the cranial operation of decompression and drainage, then this latter procedure should be performed with no hesitancy as the best and only means of obtaining the greatest ultimate improvement in these selected babies. The use of repeated lumbar punctures and thus the drainage of the intracranial hemorrhage and excess cerebrospinal fluid in selected babies should always be attempted as advocated by Doctor J. B. Sidbury, Wilmington, N. C.

If the left subtemporal decompression and drainage had not been performed upon this patient, it is very probable that the weakness of the right arm and right leg would have progressed into one of paralysis, the general convulsive seizures have become more severe and more frequent, and the death of the child possible or—in many respects even a greater misfortune and calamity, the survival of a child who later becomes the typical spastic hemiplegic with or without convulsive seizures and mentally impaired—a condition than which nothing is worse.

Case 133.—Acute severe brain injury in a newborn baby associated with cortical, supracortical and subtentorial hemorrhages and with high intracranial pressure. Bilateral decompression and drainage. Death; autopsy.

No. 575.—Baby Y. Forty-seven hours. White. U. S.

Admitted May 6, 1916—47 hours after birth. Polyclinic Hospital. Referred by Doctors R. T. Frank and R. Ottenberg.

Operations May 6, 1916—2 hours after admission. Bilateral subtemporal decompression and drainage.

Died May 7, 1916—21 hours after operation.

Family history negative. *Personal history* negative.

Present Illness.—First baby, full term, difficult labor requiring high forceps; head presentation; weight—8 lbs. Great difficulty in resuscitation on account of the extreme cyanosis. Several contusions of the scalp over the occipital area observed. Child very drowsy and did not cry, but it was considered normal until 4 hours after birth, when localized convulsive twitchings began in the right arm, then the right leg and the entire right side of the body; apparently no loss of consciousness; this twitching of the right side of the body lasted for two or three minutes and then a second one occurred—8 minutes later. During the following six hours, twenty of these convulsive twitchings of the right side of the body occurred, and then ten hours after birth a general convulsion appeared and continued for 6 minutes; two hours later, it was noticed that the left arm and left side of face were paralyzed and that the left leg was weak. The general convulsions continued until 43 hours after birth—always beginning in the right arm and the right leg and then becoming general; the paralysis of the right arm and of the right side of face persisted.

Examination at consultation (47 hours after birth and injury).—Temperature, 104°; pulse, 130; respiration, 42. Well-formed baby; semiconscious. Entire left side of body paralyzed. Slight twitchings of the right facial muscles and of the fingers of the right hand. Contusion and ecchymosis of the scalp overlying the occipital area. No bleeding from nose, mouth or ears. Both fontanelles tense and bulging; slight pulsation palpable. Pupils slightly enlarged and react to light sluggishly, right possibly larger than left. Reflexes all very much depressed; suggestive double Babinski; abdominal reflexes absent. Fundi—retinal veins widely dilated; edematous blurring of the nasal halves and temporal margins of the optic disks but no measurable papilledema. Lumbar puncture—bloody cerebrospinal fluid under increased pressure (approximately 14 mm.).

Treatment.—The presence of the blood in the cerebrospinal fluid and under increased pressure, associated with the convulsive seizures and the left hemiplegia, made the operation of cranial decompression and drainage advisable in the hope that a recovery of life might be obtained and if so, then the greatest ultimate recovery and improvement by the immediate drainage of the intracranial hemorrhage and a permanent lowering of the increased intracranial pressure; the fact, however, that the general condition of the child was very poor and practically moribund from the possible complication of medullary edema made the operation of doubtful value, and yet it was considered as giving the child its only chance of recovery (an opinion now realized to be a mistaken one, and it would have been better judgment not to have operated in the hope that the child itself might recover from this extreme condition, so that the operation could later be safely performed; as in older children and adults, the condition of medullary edema indicates the early death of the patient—operation or no operation). Before operation, the right pupil became widely dilated and the left pupil markedly contracted.

Operations (49 hours after birth).—Left and right subtemporal decompression and drainage (no anesthesia being necessary); first, left decompression: usual vertical incision, bone and newly formed bone removed, and no complications. Dura very tense and bulging, and upon incising it blood-tinged cerebrospinal fluid spurted under high pressure; the underlying edematous cortex protruded and bulged under such high pressure that a small rupture occurred below the Sylvian fissure; throughout the cortex were numerous punctate hemorrhages. No pulsation visible. Owing to this extreme intradural pressure, it was decided to perform immediately a right subtemporal decompression. Usual closure with 2 drains of rubber tissue inserted. Second, right subtemporal decompression and drainage: usual vertical incision, newly formed bone of underlying vault removed, and no complications. Dura very tense and bluish, and upon incising it a dark currant-jelly supracortical clot welled through dural opening; almost 3 ounces of this clot were evacuated, exposing a very edematous and hemorrhagic cortex which bulged under high pressure. Slight pulsation of cortex now visible. Usual closure with 2 drains of rubber tissue inserted. Duration, 50 minutes.

Post-operative Notes.—The condition of the baby was apparently not worse than before the operation; profuse drainage of blood and cerebrospinal fluid saturated the dressings, requiring their change within 8 hours; it was then observed that the child could move the left arm, which had been paralyzed before the operation. The temperature, however, remained around 103°, while the pulse did not descend lower than 140, and the respiration below 48. Fourteen hours after the operation, the general condition of the child became worse in that the temperature ascended to 105.4, the pulse- and respiration-rates to 156 and 64, respectively, and finally becoming imperceptible until the child died—a death typical of medullary edema, 21 hours after operation.

Autopsy.—In the tissues of the scalp over the median portion of the occipital bone was much free blood. No fracture of the skull ascertained, but the longitudinal sinus underlying the junction of the parietal bone with the occipital bone was torn—most probably due to an overlapping of these bones at the time of birth and the resulting tear of the underlying sinus. Over the cortex of the posterior portions of both cerebral hemispheres and much more over the right one, was a layer of supracortical hemorrhage, 1 cm. in thickness and extending forward to the right decompression opening but not within 2 inches of the left decompression opening. Beneath the tentorium and about the medulla and cerebellum was a large amount of this same currant-jelly clot which had compressed directly the medulla and therefore the early signs of medullary edema and death. Numerous punctate hemorrhages throughout the cortex of both hemispheres, but no cortical laceration except at the site of the left decompression. Ventricles negative.

Remarks.—If this patient could have been operated upon several hours earlier and before the definite signs of an acute medullary edema had appeared, it is possible that a recovery of life might have been obtained, and yet with the extensive subtentorial hemorrhage as disclosed at operation and causing a direct compression of the medulla itself, it is very doubtful if this patient could have recovered under any circumstances and under any treatment. The early onset of medullary edema was undoubtedly due to this extensive subtentorial hemorrhage—the most serious of all intracranial lesions, especially when associated with an extensive cerebral edema. It is now realized that in patients having brain injuries, either at the time of birth or later in life, and the condition of medullary edema resulting from high pressure has occurred, then these patients, with but very rare exceptions, all die—no matter what the treatment, with and without operation, and it is a mistake of judgment to advise any cranial operation upon these patients in this late stage in the belief that they are being afforded a chance of recovery; they are beyond recovery once the condition of medullary edema becomes definitely established, and it is only in those patients who are entering into the stage of medullary edema that an operation may afford in a small percentage of them a definite chance of recovery.

Owing to the compensatory lessening of the increased intracranial pressure in these newborn babies by means of a separation of the lines of suture of the skull and the elasticity of the dura itself and the bulging fontanelles, the intracranial pressure as registered by the ophthalmoscope and the spinal mercurial manometer at lumbar puncture is always less than occurs in similar cases occurring in older children and in adults, in whom the lines of suture are united, the fontanelles closed and the dura inelastic. It is for this reason that the pressure of the cerebrospinal fluid in this patient did not register higher than approximately 14 mm. and the ophthalmoscope disclosed only an edematous blurring of the nasal halves and temporal margins of the optic disks, but no measurable swelling to the extent of a papilledema—let alone the more advanced degree of "choked disks"—this latter condition being a most rare observation in patients having brain injuries and due to the fact that these patients usually die from the high intracranial pressure before the condition of "choked disks" can be produced, unless the increasing intracranial pressure is a slow and gradual one, as may result from hemorrhage of the middle meningeal artery and causing a huge extradural clot to be formed; in this manner the increasing intracranial pressure would be similar to that caused by tumor formation, only much more rapid when due to hemorrhage. If the ventricles, however, should be blocked and a mild internal hydrocephalus be produced, then the condition of "choked disks" is very easily and very early produced; fortunately, this ventricular blockage in traumatic cases is rare.

It would have been better surgical judgment if the first decompression had been performed upon the right side of the head rather than upon the left side, and the clinical signs of paralysis of the left side of the body and the larger right pupil indicated that the pressure over the right cerebral cortex was greater than over the left cerebral cortex; that is, the greater pressure over the right cerebral cortex was producing the paralytic dilatation of the ipsolateral right pupil and the paralysis of the left side of the body, whereas the less pressure of the left cerebral cortex was causing an irritative constriction of the ipsolateral left pupil and the irritative convulsive seizures of the Jacksonian type of the right side of the body. If the right subtemporal decompression had been performed first and the supracortical clot evacuated, it would then have been possible to have performed the left decompression without the operative damage of rupture of the underlying cortex due to the extreme intradural pressure.

CASE 134.—Acute severe brain injury in a newborn baby associated with an increased intracranial pressure due to subdural, supracortical and subtentorial hemorrhages and cerebral edema. Bilateral decompression and drainage. Death. Autopsy.

No. 1048.—Ninomiya. Six hours. Yellow. Japanese. U. S.

Admitted (born December 4, 1918, 12 noon), Audubon Hospital. Referred by Doctor E. A. Drummond.

Operations: 1st, December 5, 1918 (10 P.M.)—34 hours after birth. Right subtemporal decompression and drainage. 2nd, December 6, 1918 (11 A.M.) —13 hours after first operation. Left subtemporal decompression and drainage.

Died December 9, 1918—65 hours after second operation.

Family history negative.

Personal History.—Patient is first child, full-term baby, difficult labor but no instruments used; mother was given pituitrin, causing a precipitant labor; difficulty in resuscitating child, otherwise no abnormalities noticed. Four hours after birth, slight twitchings of the left side of the face began and these gradually extended to include the left arm and the left leg; no definite weakness of the left side of the body observed at this time.

Examination in consultation with Doctor Drummond, December 4, 1918, 6 P.M. (6 hours after birth).—Temperature, 101.4 °; pulse, 140; respiration, 38. Fairly well-developed and nourished Japanese baby girl. During the examination, a convulsive seizure occurred in that muscular twitchings of the left side of face began, then the left arm and the left leg jerked spasmodically and finally the entire right side of the body twitched—the whole attack not lasting more than one minute. A large fluctuating hematoma of 2 inches in diameter and very tense, extended over the right parieto-occipital area; otherwise no external evidence of head injury. Anterior fontanelle slightly bulging and tense. Both legs slightly stiff, but no weakness of the arms and legs ascertained. Pupils equal and react to light normally. Reflexes—patellar exaggerated, left possibly greater than right; no ankle clonus but suggestive double Babinski; abdominal reflexes absent. Fundi—retinal veins enlarged; nasal half of right optic disk and nasal margin of left optic disk blurred by edema. Lumbar puncture—blood-tinged cerebrospinal fluid under slightly increased pressure (9 mm.).

Treatment.—In the hope that the slightly increased intracranial pressure due to subdural hemorrhage and cerebral edema could be "taken care of" by the natural means of absorption and also, if the dura were torn, by the hemorrhage and cerebrospinal fluid escaping through the vault to form the hematoma of the right parieto-occipital area, it was considered better judgment to treat the child expectantly in the hope that the convulsive twitches would quickly cease and the increased intracranial pressure be lowered. (It would have been better surgical judgment to have performed either repeated lumbar punctures and spinal drainage or the operation of subtemporal decompression and drainage at this time.)

Examination (December 5, 1918 (9 P.M.)—33 hours after birth).—Temperature, 102°; pulse, 148; respiration, 42. Child is in a weaker condition than at preceding examination due to inability to take and retain its feedings. Convulsive twitches have continued—occurring at least once an hour and always beginning on the left side of face, particularly the orbicularis muscles of the left eye which contract spasmodically, then the twitching of the left side of the mouth begins, extending to the left arm and the left leg, to be followed by slight contractions of the right side of the body; during the latter part of these convulsive twitchings, the child frequently vomits. Definite weakness apparently of the left arm and left leg, and the left side of the face is less active. Pupils—right slightly larger than left but reaction to light is normal. Reflexes—patellar exaggerated, left more than right; no ankle clonus but double Babinski; abdominal reflexes absent. Fundi—retinal veins full; nasal half and temporal margin of right optic disk and temporal half of left optic disk obscured by edema; no measurable swelling of beginning papilledema of the right optic disk could be registered. Lumbar puncture—bloody cerebrospinal fluid under increased pressure (10 mm.). Anterior fontanelle tense and bulges slightly. The hematoma over the right parieto-occipital area remains the same size, but it is very tense.

Treatment.—The signs of an increasing intracranial pressure associated with a gradually progressive weakness of the child owing to its lack of nourishment made imperative an immediate mechanical relief of the increased intracranial pressure by means of a subtemporal decompression and drainage as the only hope of the child to recover life and then to approximate a normal child.

First Operation (34 hours after birth).—Right subtemporal decompression and drainage (primary anesthesia only being used): usual vertical incision, bone removed, and no complications; the Doyen perforator and burr were not used as the squamo-parietal suture could be incised by the scalpel and the rongeurs easily inserted beneath the bone edge—thus facilitating the removal of bone. Upon incising the dura which was very tense, bloody cerebrospinal fluid spurted to a height of 1 inch; upon enlarging this dural opening, the tense underlying cortex protruded and ruptured for a distance of 1 cm., owing to the high cerebral tension. The cortex contained so many punctate hemorrhages that it had the appearance almost of liver tissue. Much subdural and subarachnoid hemorrhage escaped, permitting the cortex to pulsate feebly. The inner surface of the dura was lined by a layer of clotted blood. Usual closure with 2 drains of rubber tissue inserted. Duration, 28 minutes.

Post-operative Notes.—Child did not have a convulsive twitch until 9 hours after this operation, when they began this time on the right side of the face, particularly in the right orbicularis muscles, and then extended into the right arm and the right leg, but no twitchings of the left side of the body at all. Profuse discharge of bloody cerebrospinal fluid into the dressings of the right subtemporal decompression and drainage; the decompression area, however, remained tense and bulging and no pulsation was visible. At an examination eleven hours after operation, the left pupil was slightly larger than the right and the right reflexes were now more active than the left; the fundi, however, disclosed the nasal halves of both optic disks as being blurred by edema. The anterior fontanelle was again tense and bulging. It was therefore considered advisable to perform a left subtemporal decompression and drainage to lessen this increasing intracranial pressure, particularly over the left hemisphere; the child had become definitely weaker during the last 12 hours.

Second Operation (13 hours after first operation).—Left subtemporal decompression and drainage: usual incision, bone removed, and no complications; the squamo-parietal suture was again separated by the scalpel and the rongeurs easily inserted for the removal of the bone. Dura rather tense and bluish; upon incising it, blood-tinged cerebrospinal fluid welled out, exposing a tense cortex over which there was a thin film of subarachnoid hemorrhage. The cortex tended to protrude but did not rupture. The arachnoid was incised, allowing dark free blood to escape. No cortical lacerations or hemorrhage visible. The under surface of the dura was covered by a layer of blood—similar to the condition of pachymeningitis hemorrhagica interna. Usual closure with 2 drains of rubber tissue inserted. Duration, 26 minutes.

Post-operative Notes.—Owing to the child refusing to take its nourishment, it became progressively weaker; an intravenous saline was given and repeated nutrient rectal enemata, but the general condition of the child did not improve; it made no effort to swallow. The blood-pressure gradually descended to 100 and below, and the child finally died, apparently of general exhaustion, 65 hours after operation.

Autopsy.—No fracture of the skull ascertained. Small tear through the right parieto-occipital line of suture underlying the hematoma, which had formed by the tearing of a vessel running from the bone to the underlying sinus; the dura itself had not been ruptured. Over both cerebral hemispheres was a thin film of subdural and subarachnoid hemorrhage—that is, a supracortical hemorrhage; a layer of clotted blood was adherent to the under surface of the dura. With the exception of the operative laceration of the cortex underlying the right subtemporal decompression, there were no lacerations or extensive cortical hemorrhages ascertained. Beneath the tentorium, there was a large amount of subdural hemorrhage and cerebrospinal fluid; cerebellum and medulla were themselves of normal appearance. Ventricles negative.

Remarks.—The progress of this patient was a most interesting one; naturally the operation of decompression and drainage should have been performed immediately after the onset of the convulsions following the first examination, 6 hours after birth; it was hoped that the hematoma was connected with the intracranial cavity and in this manner the intracranial hemorrhage and edema could be sufficiently drained together with the natural means of absorption, so that a cranial operation would be avoided. In this manner, the ideal period for the operation was allowed to pass, the child becoming weaker and weaker from lack of nourishment and together with the severe cerebral and subtentorial hemorrhage, an early death occurred. Owing to the extensive multiple hemorrhages throughout the cortex, it is doubtful whether this child could have become normal, and yet this same acute condition has been frequently observed in adults following cranial injuries and many of them returned to their former normality; in children, it would seem that they would have a still greater chance of approximating normality than adults following brain injuries, as the nerve cells in babies are less highly developed than they are in later life.

The bilateral spasticity, and particularly of the legs, is always a bad prognostic sign in these acute cases of brain injuries, not only in babies and children, but also in adults; post-mortem examinations of these patients usually reveal lesions at the base of the skull, and particlularly of the subtentorial region—most probably due to a compression of the pyramidal tracts themselves and thus the great danger of an associated medullary compression and its resulting medullary edema. Few of these patients having bilateral spasticity within 12 hours after the brain injury recover unless the increased intracranial pressure is early relieved.

The double Babinski as elicited in this patient is naturally of little or no significance, as this reflex is considered normal for babies; when associated, as it was in this child, with exaggerated patellar reflexes and especially unequal ones, then their presence may be considered confirmatory.

It was interesting to observe the increasing intracranial pressure as registered by the spinal mercurial manometer; 6 hours after birth, the pressure of the cerebrospinal fluid at lumbar puncture was 9 mm., whereas at the next test, 27 hours later, the pressure was registered by the spinal mercurial manometer as 10 mm. The normal pressure for newborn babies is 4 to 7 mm. and thus we could state with accuracy that the intracranial pressure was definitely increased. Naturally, there were the other signs of an increasing intracranial pressure, as revealed in the ophthalmoscopic examinations of the fundi, the bulging and tenseness of the fontanelles, together with the signs of cortical irritation as disclosed by the convulsive twitchings.

The beneficial effect of the right subtemporal decompression and drainage was shown in many ways; the convulsive seizures ceased for a period of 9 hours following the operation, and when they did begin they were limited to the right side of the body—that is, to the side of the body controlled by the left hemisphere, which had not been satisfactorily decompressed and its cortical irritation lessened; similarly, the right pupil which had been dilated, due to the paralytic effect of greater compression over the ipsolateral cerebral cortex, now became of normal size following the operation, and the left pupil gradually enlarged as ascertained at the next examination, 11 hours after the operation, indicating that the left cerebral cortex was now being compressed more than the right; the improvement of the reflexes and the change in their inequality to the opposite side of the body, together with the disappearance of the weakness of the left side of the body, is very impressive.

Only the slightest amount of ether was administered—in fact, only a "whiff" of it was given while the scalp was incised, and then when the dura was opened so that the child would not struggle and cause an increase of the cerebral tension. It is surprising how little anesthesia is required for cranial operations in babies under 3 months of age; besides, it is a dangerous factor and should be avoided as much as possible.

The layer of free blood adherent to the inner surface of the dura overlying both cerebral hemispheres shows the first stage of the organization of this supracortical blood-clot which becomes firmly adherent to the dura and giving the dura later a thickened, fibrous, whitish appearance—becoming a firm inelastic membrane which does not expand and thus diminishes the lessening of the intracranial pressure which would otherwise be afforded by the fontanelles; these latter cannot bulge and protrude owing to this fibrous thickening of the dura and thus the increased intracranial pressure is not lessened markedly, so that the development of spastic paralysis and mental impairment is later possible in these patients following a supracortical hemorrhage at the time of birth, unless early relieved mechanically by means of a subtemporal decompression and drainage as was attempted in this patient; in this case, however, the intracranial damage was so great, particularly the subtentorial compression and also the extreme general weakness of the child due to insufficient nourishment, that a recovery of life itself could not be obtained; it is unfortunate, however, that an earlier operative attempt to lessen the increased intracranial pressure was not made, as the child might then have been enabled to swallow and to retain its nourishment.

B. Acute Brain Injuries in Children.—In children under twelve years of age, cranial injuries may be of comparatively trivial character, and yet the most serious intracranial lesions often result—with and without a fracture of the skull. In these patients, however, a fracture of the vault and even of the base occurs much more easily than in adults, and the relative unimportance of the fracture of the skull in brain injuries is not illustrated better than in a study of these patients.

Not only do children withstand better the immediate effects of the cranial injury and especially the severity of the initial shock in that their reaction is a more vigorous one and thus assuring a higher percentage of immediate recovery of life, but it seems that the cardiac and respiratory centres in the medulla are more resistant and their circulatory mechanism more adaptable to sudden increases of the intracranial pressure; it is for this latter reason that the expectant palliative treatment can be used successfully in a larger percentage of children having brain injuries both as to the immediate recovery of life and to the future normality than is possible in adults in whom not only is the initial shock a most serious factor, but the sudden increase of the intracranial pressure is an only too frequent cause for early medullary complications of compresson, and even medullary edema itself. In this series of brain injuries in children under 12 years of age, the expectant palliative method of treatment is alone sufficient and eminently satisfactory in over three-fourths of these patients, whereas the operative treatment to lower a high intracranial pressure—whether due to hemorrhage or excess of cerebrospinal fluid—by means of a subtemporal decompression and drainage, is only indicated in about one-fourth of the total number of these patients; naturally, and it has been repeated a number of times in this book, all depressed fractures of the vault should be elevated (and this is more frequently possible than in adults) or removed —for fear of future complications and chiefly that of cortical irritation with its resulting emotional instability and even epileptiform seizures.

Cranial injuries, apparently of a very trivial character and of such slight importance at the time of the "bump," may cause an intracranial lesion of the greatest danger—not only to the immediate life of the child, but also in its remote effects, later in life, upon the normal development—mentally, emotionally and physically. The following case-history is instructive for several reasons:

On the afternoon of April the 10th, 1917, a little girl of nine years of age was returning home from school with her governess; while crossing a street six blocks from her home, she stumbled, bumping the left side of her head against the pavement; she jumped up without the assistance of her companion, and although the tears came to her eyes, yet she did not cry; she continued to walk to her home, ate her supper two hours later, and then went to bed at nine o'clock. The bump was such a trivial one that neither the child nor the governess thought of mentioning it to the mother. At four o'clock the next morning (twelve hours after the fall), the child became restless and then vomited, but did not complain of its head at that time; four hours later, just before breakfast, it again vomited, and now the child complained of a "beating" headache, especially over the left side of the head. Doctor W. B. Hoag examined the child two hours later to find only a slight tenderness over the left posterior parietal area (the site of the bump), and exaggeration of the deep reflexes—the right being possibly greater than the left; the pulse-rate was 90; there was no ecchymosis nor bleeding from the ear. At a consultation three hours later, the same signs were observed and a lessened activity of the right abdominal skin reflexes; the ophthalmoscopic examination of the fundi was negative; the child complained of a general headache and some dizziness upon raising the head; she was active mentally—so much so that she cried bitterly when we suggested her removal to a hospital for a four days' observation. She was admitted to the Polyclinic Hospital at four o'clock in the afternoon (just 24 hours after the injury); her pulse-rate at this time was 82. A Röntgen-ray picture was now taken by Doctor G. W. Welton, revealing "an irregular line of fracture extending from the occipital protuberance forward to the left squamous bone, but not down to the base of the skull" (Fig. 155). The measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer registered 16 mm. (normal 5–9 mm.); the fluid was clear. During the night the pulse gradually descended to 76 at midnight, 66 at four o'clock in the morning and at ten o'clock the pulse-rate was 58; the child had become alternately restless and drowsy—would awaken with a cry, and even scream from the intensity of the headache, and then quickly lapse into a mildly stuporous condition from which she could easily be aroused; there was now a definite inequality of the exaggeration of the deep reflexes—the right being markedly increased over the left to the degree of both a right patellar and right ankle clonus, but no Babinski reflex could be elicited; the right abdominal reflexes were now found to be entirely abolished; the right-hand grasp was possibly weaker than the left, but there was no apparent weakness of the right side of the face or of right leg; an impaired sensation could not be elicited. An ophthalmosc examination at this time revealed a marked dilatation of the retinal and an edematous blurring of the nasal halves and temporal margi the optic disks—possibly greater in the left than in the right eye; was, however, no measurable papillœdema. Speech was not impaire was there present any astereognosis. No reduction of the visual

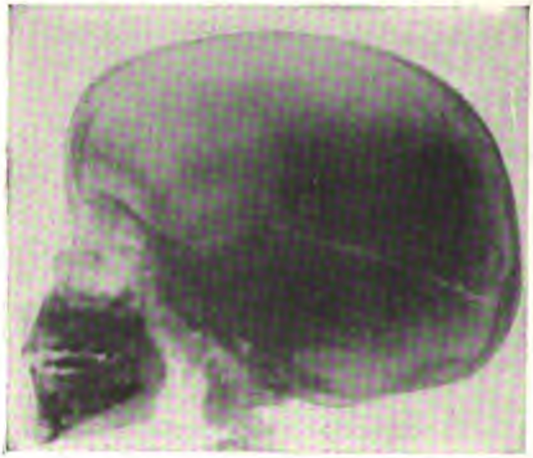

Fig. 155.—Lenore. Extensive fracture of the vault in a little girl of eleven years of age. No complaints following a trivial "bump" on the head until eighteen hours afterward; then severe headache, vomiting and definite signs of an increased intracranial pressure as shown by the ophthalmoscope and the spinal mercurial manometer. Operation performed twenty-four hours after injury revealed a large extradural hemorrhage as indicated in Fig. 157. Recovery excellent. Last examination, May 12, 1919—twenty-five months later—no complaints.

could be ascertained and an homonymous hemianopsia was not present. In order to relieve the increased intracranial pressure, whether due to cerebral edema or to hemorrhage, a rather posterior left subtemporal decompression was performed forty-four hours after the bump (Fig. 156); a small line of fracture extended obliquely downward through the squamous bone, but did not reach its base—therefore, no ecchymosis about the ear nor bleeding from the external auditory canal; upon rongeuring away the squamous bone lying beneath the temporal muscle, an extradural currant-jelly clot of the thickness of 1–1½ inches welled up through the bony opening (Fig. 157); as much as four ounces of clotted hemorrhage were removed with a spoon spatula, allowing the underlying compressed dura and brain to rise; the cavity extended upward to the longitudinal sinus and backward to the tentorium; a small amount of cerebral tissue was found in the upper portion of the clot, so that the dura had undoubtedly been torn in that area; the posterior branch of the middle meningeal artery had also been torn—the usual source of this type of hemorrhage. As the dura itself was now no longer under tension and the underlying cerebral convolutions could be clearly observed, it was thought to be better surgical judgment not to open the dura in this case. The temporal muscle and fascia were now sutured and then the scalp—two rubber tissue drains having been inserted extradurally. The convalescence was uneventful; pulse-rate became 76 upon the first day post-operative and the other signs noted above quickly faded away so that the child made an excellent recovery. She was discharged from the hospital upon the tenth day post-operative. Last examination (May 12, 1919—25 months after injury).—Negative; no complaints.

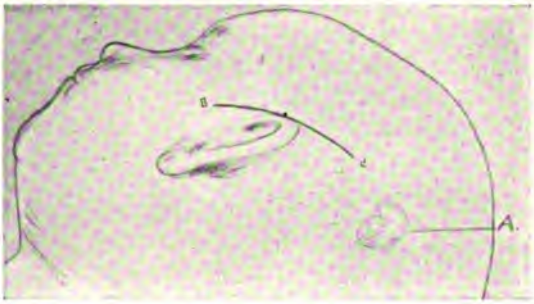

Fig. 156.—Leonore. Shows the posterior oblique incision (B-C) used at operation upon this patient. The slight bruise of the "bump" is observed at (A).

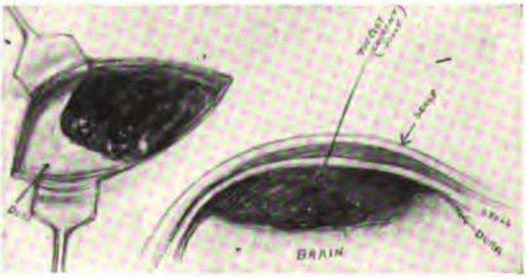

Fig. 157.—Lenore. Two views of the extra dural hemorrhage exposed and removed at operation upon this patient. Note the marked depression of underlying brain, and yet the clinical signs were most vague within twenty-four hours after injury; apparently only a trivial "bump" upon the head. The röntgenogram illustrates the advisability of having all head injuries —no matter how trivial—examined by the X-ray.

The main points of this case have been described in detail in order to illustrate the apparent triviality of the initial symptoms and signs of many brain injuries, with or without a fracture of the skull. Similar cases of an acute intracranial lesion occur not so infrequently but that we should always be most careful in our examinations of patients having a history of a recent head injury of even the most trivial character, and especially is this true of children, who withstand the effects of brain injuries much better than adults. There are several tests that should always be employed and repeatedly performed if necessary upon these patients, not only to locate the site of the brain injury but far more important to ascertain the presence or not of an increased intracranial pressure, whether that pressure is due to intracranial hemorrhage or to cerebral edema. As is well known, the fracture in these patients (if we exclude depressed fractures of the vault which should always be elevated or removed) is possibly the most unimportant part to be considered in the treatment, whereas the presence of a marked increase of the intracranial pressure, with or without fracture of the skull, should immediately cause the patient to be withdrawn from that large group of patients properly treated by the expectant palliative method, and the advisability of an early operative procedure to relieve the increased intracranial pressure should be considered.

A. *Acute cranial injuries of varying degree; no increase of the intracranial pressure; no operation. Excellent recovery.*

Case 135.—Acute cranial injury associated with cerebral concussion and with mild shock; no signs of an increased intracranial pressure. Expectant palliative treatment. Excellent recovery.

No. 1012.—Robert. Thirty-three months. White. U. S.

Admission August 20, 1918. Polyclinic Hospital. Referred by Doctor John A. Bodine.

Discharged August 22, 1918—2 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While playing upon a doorstep, child fell headlong to the brick pavement, a distance of 5 feet; loss of consciousness for 10 minutes; carried to the hospital by a passerby.

Examination upon admission (20 minutes after injury).—Temperature, 98.2°; pulse, 130; respiration, 28; blood-pressure, 102. Semiconscious but could be aroused, when he would cry intermittently and then fall asleep. Contusion of scalp over the right parietal area; bimanual examination was negative. No bleeding from nose, mouth or ears; no orbital or mastoid ecchymosis. No paralyses elicited. Pupils dilated and react sluggishly to light. Reflexes: patellar—depressed but equal; tendency to a double Babinski; abdominal reflexes absent. On account of the severity of the shock, no further examination was made at this time.

Treatment: for shock, especially external warmth, and the routine expectant palliative treatment. Within one hour, the general condition had so improved that a more thorough examination was possible.

Examination (2 hours after admission).—Temperature, 99.2°; pulse, 96; respiration, 28; blood-pressure, 110. Drowsy but can answer questions perfectly. Otoscopic examination negative. Pupils small, equal and react normally. Reflexes—patellar slightly exaggerated but equal; no ankle clonus but a bilateral Babinski; abdominal reflexes depressed but equal.

Fundi—retinal veins slightly enlarged; no edematous blurring of the margins of the optic disks. Lumbar puncture—clear cerebrospinal fluid under normal pressure (8 mm.). X-ray (Doctor G. W. Welton)—"negative for fracture of the skull."

Treatment.—Expectant palliative continued. Child improved so rapidly that by the following morning he was sitting up in bed with no complaints; slight tenderness of the bruise of the scalp. Pupils negative. Reflexes negative. Fundi—possibly a slight dilatation of the retinal veins; otherwise normal.

Examination at discharge (2 days after admission).—Temperature, 98.6°; pulse, 82; respiration, 24; blood-pressure, 112. No complaints and appears normal in every way. No paralyses nor impairments of sensation. Pupils negative. Reflexes negative. Fundi negative.

Examination (November 4, 1918—3 months after injury).—No complaints. Reflexes negative. Fundi negative.

Last Examination (May 16, 1919—9 months after injury).—No complaints; "As well as ever." Reflexes negative. Fundi negative.

Remarks.—This remarkable recuperative ability of children having cranial injuries is very common and especially between the ages of 2 and 12 years; in adults, the initial shock alone is a most serious complication, whereas in children of this age they may be in an extreme condition of shock immediately following the cranial injury and yet the next day they are practically well. Also the ability of these children to "take care of" a moderate increase of the intracranial pressure of hemorrhage or excess cerebrospinal fluid by the natural means of absorption is another factor to be remembered in their treatment—making operative interference a less frequent necessity in them than in adults having a similar intracranial condition.

External warmth, particularly heated blankets, is of the greatest value in overcoming the effects of the shock in these children—possibly more so than in adults. Rectal enemata of hot black coffee alternating each hour with normal saline solution is also very satisfactory, as is the codeine for restlessness; as a rule, however, drugs are of little value in children in this severe condition of initial shock—less so than in adults.

CASE 136.—Acute cranial injury with cerebral concussion and associated with severe shock and with a fracture of the vault, but no signs of an increased intracranial pressure. No operation. Excellent recovery.

No. 252.—Charles. Six years. White. School. U. S.

Admission May 30, 1915, Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Discharged June 5, 1915—6 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While playing in the street, child was struck by an automobile and rolled along the cement for a distance of 20 feet; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (35 minutes after injury).—Temperature, 97.8°; pulse, 136; respiration, 34; blood-pressure, 88. Unconscious;

in severe shock with extreme pallor and cold; chest, abdomen and extremities negative. Multiple contusions and abrasions about head with a tense hematoma of the size of an egg over the right forehead. No bleeding from nose, mouth or ears; ecchymosis of right orbit but not of either mastoid area. Pupils widely dilated and no reaction to light observed. Reflexes all abolished.

Treatment.—No extensive examination made on account of the extreme condition of shock, which was immediately combatted with heated blankets and hot water bottles; rectal enemata of hot black coffee (ounces 2) alternating with hot normal saline solution (ounces 2) every hour for 8 hours, with absolute quiet, in a darkened room. After 2 hours, the condition began to improve in that the temperature became almost normal and the pulse and respiration descended slightly, while the blood-pressure increased to 98; after a period of 12 hours had passed, his improved condition permitted the following examination to be made:

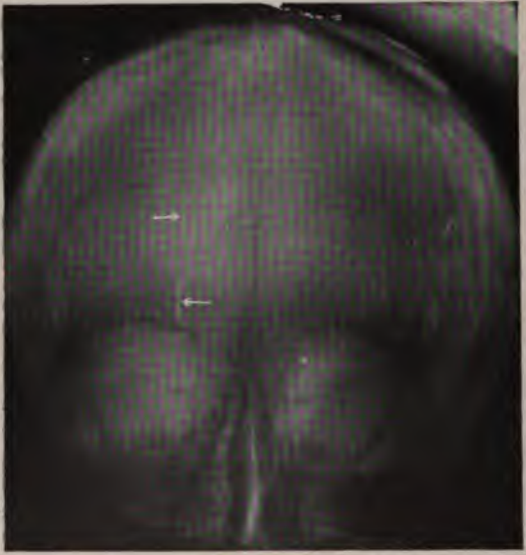

FIG. 158.—Vertical linear fracture of right half of frontal bone, extending into right orbital plate in a patient having the symptoms and signs of only concussion. The comparative unimportance of the fracture of the skull is well illustrated. Excellent recovery.

Examination (12 hours after admission).—Temperature 98.8°; pulse, 102; respiration, 28; blood-pressure, 108. Semiconscious; of better color and body warmth. External condition of head and scalp remains the same as at preceding examination. No paralysis elicited. Otoscopic examination negative. Pupils of normal size and reaction. Reflexes present and equal; no ankle clonus nor Babinski; abdominal reflexes slightly depressed but equal. Fundi negative. Lumbar puncture—clear cerebrospinal fluid under normal pressure (7 mm.). X-ray (Doctor A. J. Quimby)—"vertical line of fracture of the right half of the frontal bone extending down into orbital plate" (Fig. 158).

Treatment.—Expectant palliative continued. The improvement in the general condition was a rapid one after the first 12 hours, so that the patient could be discharged on the sixth day after the injury.

Examination at discharge (6 days after injury).—Temperature, 98.6°; pulse, 82; respiration, 24; blood-pressure, 114. No complaints except a general soreness of the entire scalp. Hematoma of right forehead is much smaller; no definite areas of tenderness. Pupils equal and react normally. Reflexes negative. Fundi negative.

Examination (June 4, 1917—24 months after injury).—No complaints; is considered "perfectly well." Reflexes negative. Fundi negative.

Last Report (February 26, 1919—45 months after injury).—No complaints; "well and strong; gets along well in school."

Remarks.—This case illustrates the remarkable ability of children to react from the extreme condition of shock following severe cranial injuries; a similar degree of initial shock in an adult would probably have been fatal. Not only can children, and especially under 12 years of age, withstand the effect of extreme shock in these injuries, but they are able to resist and "take care of" by absorption a high degree of intracranial pressure of either hemorrhage or excess cerebrospinal fluid, so that the operation of subtemporal decompression and drainage is not so frequently indicated in them as in adults having a similar condition.

The most effective anti-shock remedies are external heat by means of heated blankets being wrapped about the patient and hot water bottles to the extremities and body; internal heat and stimulation by rectal enemata of hot black coffee and hot normal saline solution; codeine and even small doses of morphia if the patient is restless; and absolute rest and quiet. The patient must not be disturbed by prolonged and extensive neurological examinations to ascertain any change of the reflexes, ophthalmoscopic examinations, and by no means a lumbar puncture in this period of severe shock; a marked increase of the intracranial pressure cannot be present in this severe stage and no treatment other than the expectant palliative one can be administered during this period—no matter what the examinations might reveal.

The relative unimportance of the fracture of the skull in these patients is well illustrated in this case-history.

CASE 137.—Acute cranial injury associated with a possible depressed fracture of the vault of the skull. Exploratory incision of the scalp—no depression ascertained Excellent recovery.

No. 240.—Annie. Fifty-four months. White. U. S.

Admission May 14, 1915, Polyclinic Hospital. Referred by Doctor C. S. Hunt.

Operation May 16, 1915—2 days after injury. Exploratory scalp incision.

Discharge May 24, 1915—8 days after scalp incision.

Family history negative.

Personal history negative.

Present Illness.—While playing in the street, child was knocked down by an automobile; unconscious for several minutes; mother brought child to the hospital on account of "lump on head."

Examination upon admission (3 hours after injury).—Temperature, 98.8°; pulse, 92; respiration, 28; blood-pressure, 108. Perfectly conscious but irritable; vomited while being taken to the ward. Tense hematoma—the size of a lemon, over the left half of the frontal bone; after very careful palpation it was decided that a depressed fracture of the underlying bone was undoubtedly present; apparent crepitus was also elicited at its posterior border. No bleeding from the nose, mouth or ears; bilateral orbital ecchymoses, but both mastoid areas were of normal appearance. Pupils slightly

in severe shock with extreme pallor and cold; chest, abdomen
negative. Multiple contusions and abrasions about b
hematoma of the size of an egg over the right forehead
nose, mouth or ears; ecchymosis of right orbit but not
Pupils widely dilated and no reaction to lig
all abolished.

Treatment.—No extensive examination made
condition of shock, which was immediately cor
and hot water bottles; rectal enemata of hot b
ing with hot normal saline solution (ounces.
absolute quiet, in a darkened room. Afte
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tory incision of scalp
n of 4 cm. made over the
ated, revealing an irregular
rlying bone was normal and no
rosis was sutured with interrupted
rubber tissue inserted beneath scalp.
ture—clear cerebrospinal fluid under

ntful recovery; all sutures removed on the

ge (8 days after scalp incision).—Temperature,
ion, 26; blood-pressure, 112. No complaints except
former hematoma. Scalp wound healed perfectly.
ts fading rapidly. Pupils equal and react normally
Fundi negative.
April 26, 1917—23 months after injury).—No complaints;
of operation"; goes to kindergarten daily. Reflexes
negative.
February 28, 1919—45 months after injury).—No com-
girl in school."

Fig. 158.—V
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the symptoms
unimportance
Excellent re

it was gross carelessness that X-ray pictures were not taken
this patient in several planes if necessary, so that this unfor-
would have been avoided; although the risk of an exploratory
is practically nil, yet when it can be avoided by careful rönt-
should certainly be at least attempted. In all doubtful cases
exploratory scalp incision is to be performed as being much the
procedure than permitting the patient to undergo the great risk of
complications, and especially in children, such as emotional instability,
epileptiform seizures, mental retardation, etc.

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Case 138.—Acute cranial injury simulating a depressed fracture of the
Exploratory incision of the scalp; no depression ascertained. Excel-
lent recovery.

No. 21.—Randolph. Four years. White. U. S.

Admitted August 1, 1913. Polyclinic Hospital. Referred by Doctor
W. S. Bainbridge.

Operation August 3, 1913—50 hours after injury. Exploratory
scalp incision.

ust 15, 1915—12 days after scalp incision.
ative.
ive.

laying upon a fire-escape, child fell to the
t; loss of consciousness for several minutes;
by a neighbor.

minutes after injury).—Tempera-
lood-pressure, 96. Conscious but
right parietal area was a boggy,
d upon palpation a distinct vertical
rked tenderness along this area; at the
etal crest the sensation of a depressed area
eeding from nose, mouth or ears; no orbital
o paralyses or impairments of sensation elicited.
and react to light sluggishly. Reflexes all depressed
ive. Fundi negative. On account of the severe con-
o prolonged examination was made.

.—Vigorous shock measures instituted. Within six hours, the
dition of the patient had so improved that he was considered
out of immediate danger.

xamination (48 hours after injury).—Temperature, 99.4°; pulse, 90; spiration, 28; blood-pressure, 108. Conscious but rather drowsy. The hematoma over the right parietal area more tense but palpation can still elicit an apparent vertical fracture with depression of its lower portion; acute local tenderness persists. Hearing negative; otoscopic examination negative. Pupils equal and react normally. Reflexes—patellar active but equal; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi—retinal veins slightly enlarged; no obscuration of the details of either optic disk. Lumbar puncture—clear cerebrospinal fluid under normal pressure (approximately 8 mm.). No X-ray picture requested in the belief that the fracture and depression of the vault did not require confirmation (a mistaken opinion and especially in a hospital having all the modern facilities for accurate work).

Treatment.—The patient no longer being in the condition of shock, an exploratory incision of the scalp overlying the depressed fracture of the right parietal bone was advised, both to elevate or remove the depressed bone and at the same time to drain the hematoma, and thus lessen the danger of the hematoma becoming infected with an extension of the infective process intracranially through the line of fracture.

Operation (50 hours after injury).—Exploratory incision of scalp (primary anesthesia): small vertical incision of 4 cm. over the right parietal crest and overlying the site of the supposed depressed area of bone. Upon retracting the scalp, the pericranium (fronto-occipital aponeurosis) was found to be torn vertically with its edges turned upward; no fracture of the underlying bone ascertained nor any depression. For fear that a depressed fracture of the inner table of the bone might be present, a small opening was made with the Doyen perforator and burr and enlarged with rongeurs to a diameter of 2 cm.; no fracture or depression of the inner table observed enlarged but equal and of normal reaction to light. Reflexes: all depressed but equal; no Babinski. Fundi negative. On account of the mild degree of shock, it was decided to postpone more careful examinations and the elevation or removal of the depressed area of the vault until the general condition of the patient was better.

Treatment.—Routine shock measures and the expectant palliative method. Within 12 hours, child seemed almost well except for the tense swelling over the left half of the frontal bone; as a depressed fracture of the underlying bone had appeared so evident, no X-ray picture was requested—a careless mistake and an inexcusable one in a hospital equipped properly.

Operation (2 days after admission).—Exploratory incision of scalp (primary ether anesthesia): small vertical incision of 4 cm. made over the hematoma; much dark gelatinous blood evacuated, revealing an irregular tear of the fronto-occipital aponeurosis; underlying bone was normal and *no* fracture of the vault ascertained. Aponeurosis was sutured with interrupted catgut. Usual closure with 2 drains of rubber tissue inserted beneath scalp. Duration, 18 minutes. Lumbar puncture—clear cerebrospinal fluid under normal pressure (8 mm.).

Post-operative Notes.—Uneventful recovery; all sutures removed on the fifth day.

Examination at discharge (8 days after scalp incision).—Temperature, 98.6°; pulse, 82; respiration, 26; blood-pressure, 112. No complaints except soreness at the site of former hematoma. Scalp wound healed perfectly. Ecchymoses of both orbits fading rapidly. Pupils equal and react normally. Reflexes negative. Fundi negative.

Examination (April 26, 1917—23 months after injury).—No complaints; "no hair along place of operation"; goes to kindergarten daily. Reflexes negative. Fundi negative.

Last Report (February 28, 1919—45 months after injury).—No complaints; "bright girl in school."

Remarks.—It was gross carelessness that X-ray pictures were not taken of the skull of this patient in several planes if necessary, so that this unfortunate mistake would have been avoided; although the risk of an exploratory scalp incision is practically nil, yet when it can be avoided by careful röntgenograms, it should certainly be at least attempted. In all doubtful cases, however, an exploratory scalp incision is to be performed as being much the safer procedure than permitting the patient to undergo the great risk of future complications, and especially in children, such as emotional instability, epileptiform seizures, mental retardation, etc.

CASE 138.—Acute cranial injury simulating a depressed fracture of the vault. Exploratory incision of the scalp; no depression ascertained. Excellent recovery.

No. 21.—Randolph. Four years. White. U. S.

Admitted August 1, 1913. Polyclinic Hospital. Referred by Doctor W. S. Bainbridge.

Operation August 3, 1913—50 hours after injury. Exploratory scalp incision.

Discharged August 15, 1915—12 days after scalp incision.

Family history negative.

Personal history negative.

Present Illness.—While playing upon a fire-escape, child fell to the ground—a distance of twenty feet; loss of consciousness for several minutes; patient was carried to the hospital by a neighbor.

Examination upon admission (15 minutes after injury).—Temperature, 97.8°; pulse, 140; respiration, 34; blood-pressure, 96. Conscious but very stuporous; in severe shock. Over right parietal area was a boggy, doughy hematoma and ecchymosis, and upon palpation a distinct vertical line of fracture was apparent; marked tenderness along this area; at the lower portion over the right parietal crest the sensation of a depressed area of bone was obtained. No bleeding from nose, mouth or ears; no orbital or mastoid ecchymoses. No paralyses or impairments of sensation elicited. Pupils equally enlarged and react to light sluggishly. Reflexes all depressed but otherwise negative. Fundi negative. On account of the severe condition of shock, no prolonged examination was made.

Treatment.—Vigorous shock measures instituted. Within six hours, the general condition of the patient had so improved that he was considered as being out of immediate danger.

Examination (48 hours after injury).—Temperature, 99.4°; pulse, 90; respiration, 28; blood-pressure, 108. Conscious but rather drowsy. The hematoma over the right parietal area more tense but palpation can still elicit an apparent vertical fracture with depression of its lower portion; acute local tenderness persists. Hearing negative; otoscopic examination negative. Pupils equal and react normally. Reflexes—patellar active but equal; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi—retinal veins slightly enlarged; no obscuration of the details of either optic disk. Lumbar puncture—clear cerebrospinal fluid under normal pressure (approximately 8 mm.). No X-ray picture requested in the belief that the fracture and depression of the vault did not require confirmation (a mistaken opinion and especially in a hospital having all the modern facilities for accurate work).

Treatment.—The patient no longer being in the condition of shock, an exploratory incision of the scalp overlying the depressed fracture of the right parietal bone was advised, both to elevate or remove the depressed bone and at the same time to drain the hematoma, and thus lessen the danger of the hematoma becoming infected with an extension of the infective process intracranially through the line of fracture.

Operation (50 hours after injury).—Exploratory incision of scalp (primary anesthesia): small vertical incision of 4 cm. over the right parietal crest and overlying the site of the supposed depressed area of bone. Upon retracting the scalp, the pericranium (fronto-occipital aponeurosis) was found to be torn vertically with its edges turned upward; no fracture of the underlying bone ascertained nor any depression. For fear that a depressed fracture of the inner table of the bone might be present, a small opening was made with the Doyen perforator and burr and enlarged with rongeurs to a diameter of 2 cm.; no fracture or depression of the inner table and the underlying dura was negative. Usual closure with 2 drains of rubber tissue inserted. Duration, 25 minutes.

Post-operative Notes.—No complications, so that the patient made an uneventful recovery.

Examination at discharge (12 days after operation).—Temperature, 98.8°; pulse, 86; respiration, 24; blood-pressure, 110. Operative wound healed *per primam*. Pupils negative. Reflexes negative. Fundi negative. No complaints and "I want to go home."

Examination (September 20, 1915—23 months after injury).—No complaints. Slight pulsation palpable at site of removal of bone. Reflexes negative. Fundi negative.

Last Examination (June 12, 1918—70 months after injury).—No complaints referable to the former cranial injury; has been going to school daily and his reports are excellent. Reflexes negative. Fundi negative.

Remarks.—The mistake illustrated in this case-history is a rather frequent one, unless X-ray pictures are taken as a routine procedure of all cranial injuries; naturally, it would be advisable to perform an exploratory operation as in this patient, even in the absence of a depressed fracture of the vault, than to overlook a depressed area of the vault and in all doubtful patients an exploratory incision should be made; careful röntgenograms should always be taken when possible and thus obviate the necessity of an exploratory scalp incision in the absence of a bony depression. The danger of future complications is very great indeed if a depressed fracture of the vault is overlooked and not elevated or removed, and no patient should be permitted to run that risk without every facility of careful examination and diagnosis having been utilized.

Whenever a fracture of the vault underlying an extensive, tense hematoma is ascertained, either by palpation or by röntgenograms, and there is a definite chance for the hematoma to become infected through the overlying bruised and contused skin, it is always wiser to incise and drain the hematoma through a small opening and through a normal portion of the skin, and thus lessen the great danger of an infective process extending through the line of fracture to produce a purulent meningitis with its great danger and many complications.

CASE 139.—Linear fractures of both tables of the vault underlying an extensive hematoma of the scalp; no signs of an increased intracranial pressure. No operation except drainage of hematoma. Excellent recovery.

No. 731.—Esther. Four years. White. U. S.

Admitted November 24, 1917—6 days after cranial injury. Polyclinic Hospital. Referred by Doctor George W. Hawley.

Operation November 26, 1917—2 days after admission. Drainage of hematoma.

Discharged November 29, 1917—9 days after scalp incision.

Family history negative.

Personal history negative.

Present Illness.—While at play, patient fell from a small go-cart, the right side of her head; no loss of consciousness and no bleeding nose, mouth or ears; no appa ts from the injury and its regular supper. The next morning, a large hematoma of almost 3 inches in diameter was present over the right parietal area—the site of the "bump"; very tense and did not fluctuate. During the next four days, the child seemed normal in every way, except for a slight drowsiness and the complaint of headache. No abnormal neurological signs of an intracranial lesion.

Examination upon admission (6 days after injury).—Temperature, 98.8°; pulse, 80; respiration, 26; blood-pressure, 116. Conscious and apparently normal both mentally and physically. Tense hematoma over right parietal area—almost 3 inches in diameter. Careful bimanual examination revealed no fracture of the underlying and adjacent bone. Pupils equal and react normally. Reflexes—patellar present and equal; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi negative except for slight dilatation of the retinal veins; no edematous blurring of the optic disks. Lumbar puncture—slight blood-tinged cerebrospinal fluid under normal pressure (7 mm.). X-ray (Doctor G. W. Welton)—"a horizontal linear fracture of right parietal bone underlying the hematoma of scalp" (Fig. 159).

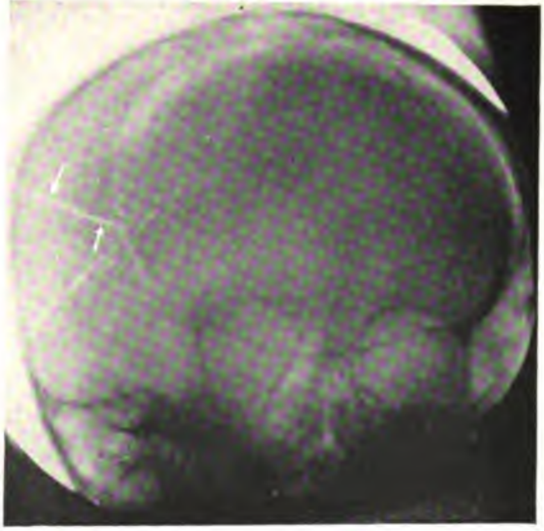

FIG. 159.—Extensive linear fracture of right parietal bone concealed by an overlying hematoma in a patient who made an excellent recovery with the expectant palliative treatment and the drainage of the hematoma. The importance of röntgenograms of all patients having head injuries is obvious for fear of later complications.

Treatment.—As the hematoma was gradually enlarging and had become much more tense during the past 48 hours, and especially in the presence of an underlying fracture of the vault but with no signs of an increased intracranial pressure, it was considered advisable to drain the hematoma through a small scalp incision and thereby afford a safe means of drainage for the intracranial hemorrhage and excess cerebrospinal fluid, but also to lessen the danger of the hematoma becoming infected and thus the great risk of a later purulent meningitis. Accordingly, the entire right half of the head was closely shaved, iodine applied and a small scalp incision of one-eighth of an inch long, made at the lower edge of the hematoma; much dark blood (almost three ounces) with cerebrospinal fluid escaped, permitting the swelling to subside entirely; no drain inserted (for fear of increasing the danger of infection). Otherwise, the routine expectant palliative treatment.

Examination (1 day after admission).—Temperature, 98.6°; pulse, 82; respiration, 24; blood-pressure, 114. Mother states that the child "is showing more interest in things"; not so drowsy and "wishes to play with her doll." Pupils equal and react normally. Reflexes negative. Fundi negative, and the retinal veins are now of normal size. Upon removing the head bandage, the hematoma had refilled but not tensely, and upon inserting a probe into the small scalp incision, about one ounce of dark syrupy blood and cerebrospinal fluid escaped so that the swelling of the hematoma again disappeared. This same treatment of drainage was used the following day and a smaller amount of dark blood and cerebrospinal fluid permitted to escape, and upon the application of a firm head bandage, it was ascertained the next day that the hematoma did not refill.

Examination at discharge (5 days after admission).—Temperature, 98.6°; pulse, 82; respiration, 26; blood-pressure, 116. No complaints and apparently normal in every way. Site of former hematoma flat and the overlying scalp is now adjacent to the vault; small scalp incision has healed *per primam*. Pupils negative. Reflexes negative. Fundi negative.

Examination (May 20, 1918—6 months after injury).—No complaints; "child is as well as ever." Reflexes negative. Fundi negative.

Last Report (March 18, 1919—16 months after injury).—(Letter from mother): "Esther is in the best of health and seems normal in every way."

Remarks.—If an X-ray picture had not been obtained in this patient, it is probable that the condition would have been considered merely as a "bump" upon the head with a resulting hematoma of large persistent character; it is also very probable that, if no complications of infection of the hematoma occurred, that this patient would have made an excellent recovery, even if the hematoma had not been drained through the scalp incision. However, the danger of infection of the hematoma in these patients is a definite factor and a distinct danger, and if it had occurred in the presence of the underlying fracture of the vault and the torn dura (since cerebrospinal fluid was present in the discharge), the result would undoubtedly have been a fatal one. If the röntgenogram had not disclosed a fracture of the underlying vault, then an operative drainage of the hematoma, either by means of a small scalp incision or aspirating needle, would have been optional and, unless the hematoma persisted for a number of days and was even enlarging on account of the tenseness, it is usually not necessary to drain it.

The presence of the fracture of the vault in this patient, and especially associated with a tear of the underlying dura, made it possible for the subdural hemorrhage and the excess cerebrospinal fluid to escape extracranially into the subcutaneous tissues of the scalp and thus, even at the risk of an infection (and this is a definite factor in patients where the overlying scalp is lacerated and contused), this patient really "decompressed" herself so that an increased intracranial pressure did not appear to a degree higher than was exhibited by the slight dilatation of the retinal veins, and the spinal mercurial manometer did not register a pressure above the normal (and it is the most accurate test). If this fracture of the vault with the laceration of the adjacent dura had not occurred, it is very probable that this amount of subdural hemorrhage and excess cerebrospinal fluid would have produc a marked increase of the intracranial pressure, and it is possible that t operation of subtemporal decompression and drainage would have b advisable, if the expectant palliative treatment was not sufficient to lo this intracranial pressure by the natural means of absorption.

CASE 140.—Acute mild brain injury associated with an extensive linear fracture of the vault; no signs of an increased intracranial pressure. No operation. Excellent recovery.

No. 552.—Constance. Ten years. White. School. U. S.

Admitted April 6, 1916. Polyclinic Hospital. Referred by Doctor J. H. Fuchsius, New Rochelle.

Discharged April 10, 1916—4 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While crossing the road, patient was knocked down by a taxicab; unconscious for several minutes. Upon recovery from the severe shock, the child was brought to the hospital in an automobile.

Examination upon admission (3 hours after injury).—Temperature, 98.8°; pulse, 92; respiration, 26; blood-pressure, 106. Conscious; in mild degree of shock. Large boggy hematoma over the entire occipital area of the vault, and especially over the left side; the overlying scalp was not bruised or lacerated. No bleeding from nose, mouth or ears; both mastoid areas ecchymotic — left more than right. Otoscopic examination negative. Careful bimanual examination negative, except for tenderness in the left mastoid and left half of the occipital bone. No paralysis nor impairment of sensation elicited. Pupils equal and react normally. Reflexes—patellar possibly depressed but equal; no ankle clonus nor Babinski; abdominal reflexes sluggish but equal. Fundi negative. Lumbar puncture—clear cerebrospinal fluid under normal pressure (8 mm.). X-ray (Doctor W. H. Stewart)—"an extensive linear fracture beginning in the posterior occipital prominence at the inferior portion and extending upward and forward across the left lambdoidal suture and ending in the posterior portion of the left parietal bone" (Fig. 160).

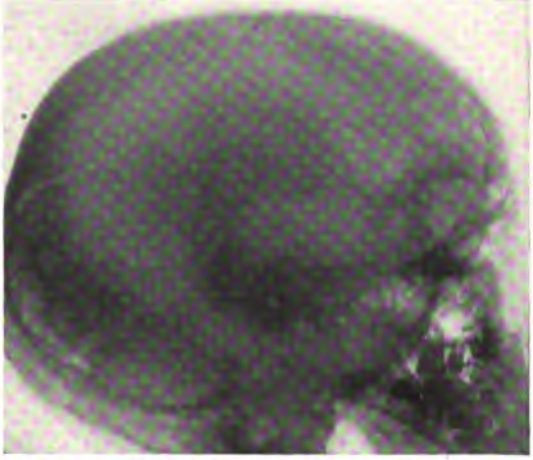

FIG. 160.—Extensive linear fracture of the posterior portion of left vault, in a patient having no signs of an increased intracranial pressure. Excellent recovery with the expectant palliative treatment.

Treatment.—Routine treatment for the mild condition of shock assocated with the usual expectant palliative method of treatment. The patient made an excellent recovery in that 6 hours later, the temperature was 99.2°, pulse 82, respiration 24, while the blood-pressure had ascended to 114. The hematoma over the back of the head did not become tense and the overlying scalp remained in an excellent condition. No signs of an increased intracranial pressure elicitable.

Examination at discharge (4 days after admission).—Temperature, 98.6°; pulse, 80; respiration, 24; blood-pressure, 116. No complaints except for soreness over the back of the head, and especially over the left side; otherwise well. Hearing normal. Pupils negative. Reflexes negative. Fundi negative.

Examination (Sept. 4, 1917—17 months after injury).—No complaints. School reports of teacher excellent. Reflexes negative. Fundi negative.

Last Report (April 24, 1919—36 months after injury).—No complaints; perfectly well.

Remarks.—Although the fracture of the vault was situated in a dangerous area, yet no complication from the underlying sinuses resulted. The line of fracture undoubtedly permitted the escape of intracranial blood and possibly cerebrospinal fluid to drain into the tissues of the overlying scalp and thus permitting a normal intracranial pressure and insuring an excellent recovery. If the skin of the adjacent scalp had been bruised and its resistance lowered, then the danger of an infective process extending to the hematoma, and even intracranially, would have been a most serious complication.

It is rather surprising that the line of fracture in this patient did not extend into the left middle ear, since this complication usually occurs when the mastoid portion of either temporal bone is fractured; the otoscopic examination is conclusive and not merely a bleeding from the ear, unless mixed with cerebrospinal fluid.

Case 141.—Acute brain injury associated with a fracture of the vault and with a small amount of subdural hemorrhage; mild signs of an increased intracranial pressure. No operation. Excellent recovery.

No. 1017.—Mary. Six years. White. School. U. S.

Admitted August 30, 1918. Polyclinic Hospital. Referred by Doctor J. A. Bodine.

Discharged September 13, 1918—14 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While playing upon a fire-escape, child fell to the ground below—a distance of 20 feet; loss of consciousness for several minutes; brought to the hospital by the mother.

Examination upon admission (2 hours after injury).—Temperature, 97.2°; pulse, 100; respiration, 30; blood-pressure, 106. Rather drowsy and in moderate shock. Extensive hematoma over the left parietal area; the overlying scalp is not damaged. No bleeding from nose, mouth or ears; no orbital or mastoid ecchymoses. No paralyses ascertained. Pupils—slightly enlarged but equal and of normal reaction to light. Reflexes: all depressed but equal; no Babinski. Fundi negative.

Treatment.—Vigorous shock measures instituted—especially heated blankets and hot water bottles, rectal enemata of hot black coffee and warm normal saline solution. The patient reacted very quickly to these measures, so that the clinical picture changed rapidly from one of shock to that o a definite intracranial lesion of mild degree.

Examination (3 hours after admission—5 hours after injury).—T perature, 100.6°; pulse, 76; respiration, 26; blood-pressure, 112. C is very drowsy—cries upon being aroused, and then becomes stuporous almost immediately. Hematoma over the left parietal area is not enlarging or becoming more tense. Left orbital ecchymosis; no mastoid discoloration. Pupils equal and react normally. Reflexes—patellar exaggerated but equal; double ankle clonus and double Babinski; abdominal reflexes depressed—left can scarcely be elicited. Fundi—retinal veins slightly enlarged; nasal margins of both optic disks blurred by edema. Lumbar puncture—bloody cerebrospinal fluid under a slightly increased pressure (8 mm.). X-ray (Doctor G. W. Welton)—"fissured oblique linear fracture through the upper portion of the left frontal bone backward into the left parietal bone; no depression" (Fig. 161).

Treatment.—Expectant palliative treatment continued, assisted by lumbar puncture with drainage of 15 c.c. of the bloody cerebrospinal fluid upon the following two days, when the pressure was only 7 mm. The patient made an excellent recovery in that after 30 hours the stupor disappeared, normal consciousness returned, and the positive signs of an intracranial lesion quickly faded away.

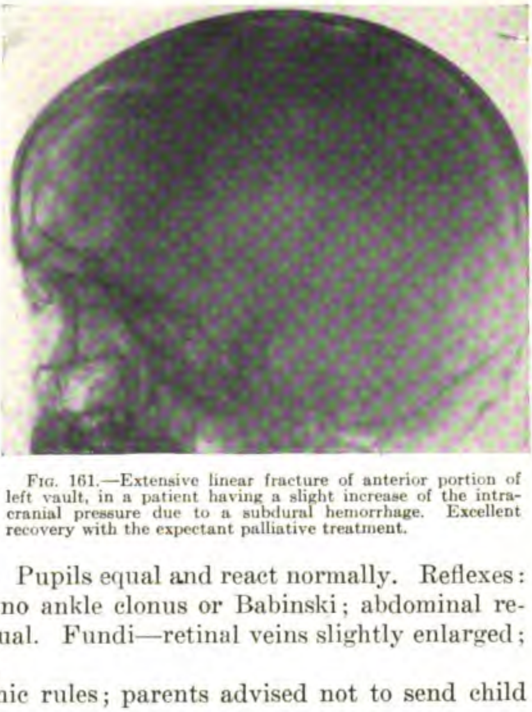

FIG. 161.—Extensive linear fracture of anterior portion of left vault, in a patient having a slight increase of the intracranial pressure due to a subdural hemorrhage. Excellent recovery with the expectant palliative treatment.

Examination at discharge (14 days after admission).—Temperature, 98.6°; pulse, 80; respiration, 24; blood-pressure, 114. No complaints except for a general soreness of the left side of head; scalp slightly boggy over that area. Hearing negative: otoscopic examination negative. Pupils equal and react normally. Reflexes: active but otherwise negative; no ankle clonus or Babinski; abdominal reflexes possibly depressed but equal. Fundi—retinal veins slightly enlarged; details of both optic disks clear.

Treatment.—General hygienic rules; parents advised not to send child to school for one year.

Last Examination (May 18, 1919—9 months after injury).—No complaints. Reflexes negative. Fundi negative.

Remarks.—The rapid recovery from the initial shock by this patient is characteristic of children, and then the signs of an intracranial lesion can be easily and safely demonstrated, but no attempt to examine the patient carefully should be made in the presence of a severe degree of shock which would be prolonged, if not increased.

Lumbar puncture drainage of the free blood in the cerebrospinal fluid in the absence of high pressure is an excellent therapeutic measure; also if the pressure is only moderately increased—not over 15 mm.—then this method of lessening the excess cerebrospinal fluid can be used, especially in children, and thus the operation of cranial decompression and drainage be avoided; in these selected patients, this method of spinal drainage may be considered a part of the expectant palliative treatment.

The presence of a linear fracture of the vault underlying the hematoma would not have been recognized had a positive röntgenogram not been obtained, and yet the treatment remains the same—fracture or no fracture. If a depression had been demonstrated, naturally its elevation or removal would have been advisable; also if the overlying scalp had been badly contused and infected, then the aspiration of a tense hematoma through a clean area of the scalp or even a small drainage incision would have been urged, in order to lessen the great danger of the hematoma becoming infected and thus the extension of the infective process through the linear fracture intracranially—the history of occasional cases of purulent meningitis following cranial injuries.

Case 142.—Acute severe cranial injury associated with a fracture of the base of the skull, but with no increase of the intracranial pressure. No operation. Excellent recovery.

No. 83.—George. Nine years. White. School. U. S.

Admitted May 14, 1914. Polyclinic Hospital. Referred by Doctor R. E. Brennan.

Discharged May 18, 1914—4 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While crossing Broadway, patient was knocked down by an automobile; unconscious for several minutes; brought to the hospital in the ambulance.

Examination upon admission (40 minutes after injury).—Temperature, 98°; pulse, 120; respiration, 26; blood-pressure, 106. Rather stuporous and drowsy but upon being aroused, the patient answers questions clearly; severe degree of shock; multiple contusions about the head, especially over the right parietal area, which is boggy and tender. Profuse bleeding from nose and left ear; small amount of cerebrospinal fluid observed in the aural discharge; otoscopic examination of right ear discloses the right tympanic membrane to be bulging and bluish. Double orbital and mastoid ecchymoses (Figs. 162 and 163). Pupils—slightly enlarged but equal and react to light sluggishly. Reflexes: all depressed but otherwise negative; no Babinski. Fundi negative. For fear of increasing the condition of shock, no further examination was made at this time.

Treatment.—Vigorous shock measures instituted and the usual routine expectant palliative treatment. Within 6 hours, the condition of the patie had so improved that the temperature was now 99°, pulse 96, respiratio 24 and the blood-pressure 118; the bleeding from both the nose and t left ear had ceased; the pupils were negative, while the reflexes were act but otherwise negative; ophthalmoscopic examination disclosed a possi enlargement of the retinal veins, but no obscuration of the details of eit optic disk; a lumbar puncture now performed permitted clear cerebrospi fluid to escape under normal pressure (approximately 8 mm.). X-ray report (Doctor A. J. Quimby)—"no fracture of the skull observed."

Treatment.—The expectant palliative treatment was continued and as the patient did not feel sick in any way and had no complaints, he insisted with the aid of his parents upon being discharged—87 hours after the injury.

Examination at discharge (4 days after admission).—Temperature, 98.8°; pulse, 80; respiration, 24; blood-pressure, 118. No complaints, except for slight dull headache and "I'm going home and away from this joint." Both orbital and mastoid ecchymoses less extensive. Hearing of both ears impaired—left more than right, and bone conduction was greater than air conduction in both ears; otoscopic examination reveals a small laceration of the upper posterior portion of the left tympanic membrane; right tympanic membrane less bulging but it is still bluish. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—retinal veins possibly enlarged but otherwise negative.

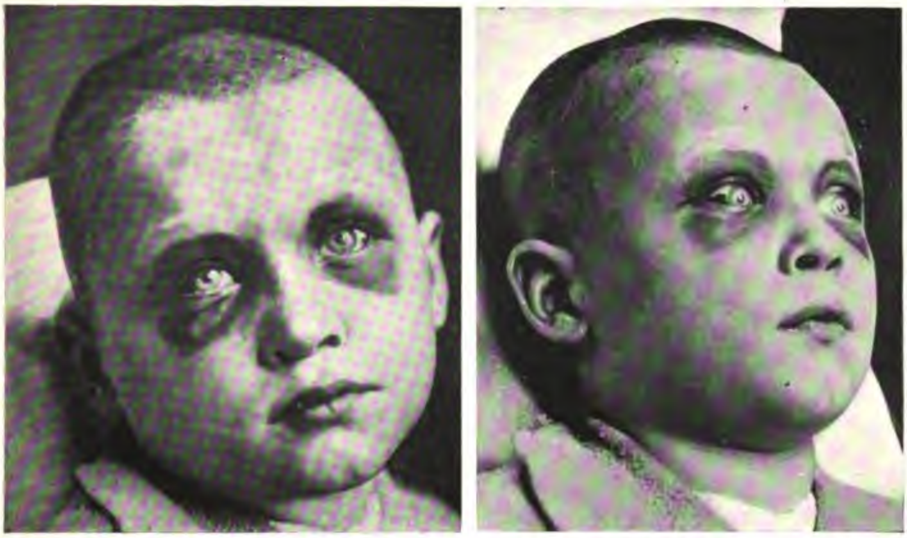

FIG. 162.—Bilateral orbital ecchymosis with right and left subconjunctival hemorrhages in a patient having a fracture of the base of the skull but no increase of the intracranial pressure. Excellent recovery with the expectant palliative treatment.

Treatment.—Parents cautioned to keep the patient in bed for at least a week and under the routine expectant palliative treatment; not to attempt to cleanse the ears or the nose; light non-stimulating diet; daily catharsis. Parents were obliged to sign the hospital blank "discharged at own request."

Examination (September 10, 1917—40 months after injury).—No complaints referable to the former head injury; "stands well in school"; no headache. Slight impairment of hearing of right ear but left ear is normal; otoscopic examination discloses a normal left tympanic membrane but a slightly thickened and retracted right tympanic membrane; bone conduction equals air conduction in right ear, whereas air conduction is greater than bone conduction in left ear (the normal condition). Reflexes negative. Fundi negative.

Last Examination (April 16, 1919—73 months after injury).—No complaints. Father states he is "just as well as if the bump on the head had not happened; he certainly fooled the doctors." Hearing the same as at preceding examination, the hearing of the right ear being slightly less acute than that of the left ear. Reflexes negative. Fundi negative.

Remarks.—The comparative unimportance of the fracture of the skull in these patients having cranial injuries, and even of the base of the skull, is well illustrated by this patient; it is undoubtedly of rather frequent occurrence to have a fracture of the skull present—more often of the vault but also of the base, and yet there are no signs of a definite intracranial lesion resulting from the cranial injury, and these latent fractures of the skull are the main pathological condition to be ascertained—usually by the X-ray in fractures of the vault or by the discharge of cerebrospinal fluid, occasionally röntgenograms, and also by the otoscope in fractures of the base of the skull. The danger of an infective process extending through the line of fracture, and particularly in fractures of the base, is a definite risk, and yet it is a slight one, unless meddlesome procedures such as the irrigation and swabbing out of the ears and nose should be attempted; in fractures of the vault, and especially in compound fractures or in some cases of tense hematomata with severe contusion of the overlying scalp—in these patients a rigid asepsis is essential and the drainage of the hematomata in selected cases.

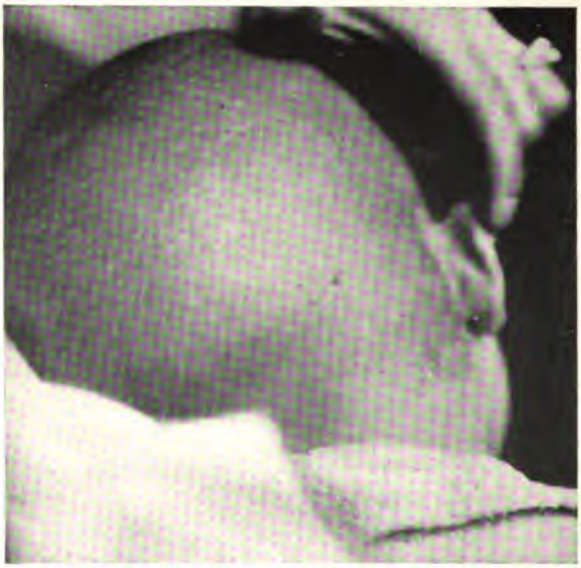

FIG. 163.—Showing right mastoid ecchymosis in a patient having a fracture of the base of the skull—the left tympanic membrane torn and permitting the escape of cerebrospinal fluid, whereas the right tympanic membrane remained intact but bluish and bulging. Excellent recovery with the expectant palliative treatment.

The rapid recovery and the uneventful convalescence of this patient i rule in children rather than the exception, in almost three-fourths o patients, and it also occurs in adults in almost two-thirds of the pati the recovery is not so rapid in the latter patients but it is usually an un ful one. These excellent recoveries are due not to the presence or al of a fracture of the skull, but to the presence or absence of a marked in of the intracranial pressure and whether the intracranial condition is hemorrhage or of edema; in either case, if the increased intracranial pressure is a high one, then it is essential and safer to lessen it by the operation of decompression and drainage, but if the increased intracranial pressure is normal or only slightly above normal, then the expectant palliative method of treatment is entirely satisfactory; repeated lumbar punctures and drainage can be used for selected patients in whom the increased intracranial pressure is a mild one. The presence or absence of a fracture of the skull—unless it is a depressed fracture of the vault—is of little value and of little importance in the treatment of these patients.

Case 143.—Acute severe cranial injury associated with multiple compound linear fractures of the vault; streptococcic infection of the cranial wound with symptoms and signs of meningeal irritation. No operation; anti-streptococcic serum administered. Excellent result.

No. 73.—Lewis. Six years. White. School. U. S.

Admitted May 4, 1914, Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Discharged May 14, 1914—10 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While playing in the street, patient was struck upon the forehead by a broom-handle, which had fallen from a distance of 4 stories; momentarily stunned, but apparently no loss of consciousness; child was able to walk to the hospital.

Examination upon admission (20 minutes after injury).—Temperature, 98.6°; pulse, 90; respiration, 28; blood-pressure, 106. Conscious and in little or no shock; "somebody hit me over the head." Just within the hairline of the median portion of the frontal bone was a lacerated wound of the scalp, one and a half inches in length and extending over the longitudinal sinus; after shaving the surrounding area and carefully cleansing the wound with soap and water, gentle probing revealed 3 distinct lines of fracture extending from the underlying area of bone; no depression of the bone ascertained; wound carefully swabbed with iodine solution and sterile dressing applied. No paralyses nor sensory impairments ascertained. No bleeding from nose, mouth or ears; no orbital nor mastoid ecchymoses. Hearing negative; otoscopic examination negative. Pupils equal and react to light normally. Reflexes negative. Fundi negative. Lumbar puncture—clear cerebrospinal fluid under normal pressure (approximately 7 mm.). X-ray (Doctor A. J. Quimby)—"multiple lines of fracture extend irregularly for several inches through the frontal and parietal bones" (Fig. 164).

Treatment.—The expectant palliative treatment in addition to the local treatment of the wound which was again dressed 8 hours later; 3 silk sutures inserted loosely and the 2 drains of rubber tissue replaced. Patient showed no ill-effects of the cranial injury and was apparently making an excellent recovery, when 30 hours later the child vomited, had a chill, and it was ascertained that the temperature had quickly ascended to 104.6°, the pulse to 96, the respiration to 30, and the blood-pressure to 114. There was definite rigidity of the neck, a positive Kernig test, and the child had become rather drowsy and stuporous; there was no change in the reflexes, but the ophthalmoscope disclosed slightly enlarged retinal veins, while the lumbar puncture removed clear cerebrospinal fluid under a slightly increased pressure (approximately 10 mm.); 16 c.c. were withdrawn; the laboratory report (Doctor Jeffries)—"14 cells per cubic mm. but no bacteria found."

Treatment.—Upon dressing the scalp wound overlying the linear fractures of the frontal bone, several drops of a thin seropurulent secretion were obtained and the report of the culture was "pure streptococci—short-chained." Wound was opened and cleansed again with iodine, lightly packed with sterile gauze and a wet bichloride (1–5000) dressing applied; a large head dressing kept saturated in the bichloride solution. Anti-streptococcic serum was immediately administered in its usual dose of 10 c.c. and within one-half hour, the temperature began to drop and 2 hours later the temperature was 102°, and the following day it was down to normal; another lumbar puncture was now performed and clear cerebrospinal fluid was found to be under normal pressure (approximately 8 mm.), while the laboratory report was only 7 cells per c.mm. The rigidity of the neck and the positive Kernig were not present, and the child became bright mentally and did not complain even of headache. The general and local condition of the child rapidly improved so that the convalescence was uneventful.

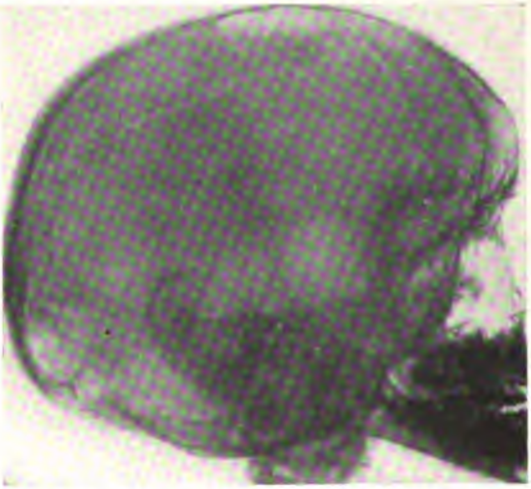

FIG. 164.—Extensive multiple linear compound fractures of frontal bone and right parietal bone, in a patient developing signs of meningeal irritation; no increased intracranial pressure. Excellent recovery with the expectant palliative treatment.

Examination at discharge (10 days after injury).—Temperature, 98.6°; pulse, 84; respiration, 24; blood-pressure, 114. No complaints; slight soreness and tenderness over the area of the former scalp wound which has now healed perfectly. Pupils equal and react to light normally. Reflexes negative. Fundi negative.

Examination (April 20, 1916—23 months after injury).—No complaints referable to the former head injury; is doing well in school. Reflexes negative. Fundi negative.

Last Report (June 12, 1919—61 months after injury).—Mother states that child does not show any ill effects of the former injury; "no differe from other boys."

Remarks.—The danger of a purulent meningitis resulting fron scalp infection in this patient, and especially in the presence of the mul fractures of the underlying bone, was very great indeed; the succe cleansing of the wound itself, however, together with the early administ of the anti-streptococcic serum undoubtedly aided the resistance of t sues to the infection, so that the signs of meningeal irritation rapidly subsided and an excellent result was obtained. It is rather puzzling that a persistent discharge of the purulent secretion did not result from the infection of the bone itself and especially at the site of the fractures; apparently the infection had not become sufficiently established before the iodine was applied.

If there had not been a rapid subsidence of the symptoms and signs of the meningeal irritation, then it would have been advisable to have performed as early as possible a subtemporal decompression and drainage and the local operation of the removal of the fractured area of bone underlying the infected scalp wound, and in this manner not only would the local source of the infection be removed, but the decompression operation and drainage would have lowered the increasing intracranial pressure and have thus permitted the brain and its meninges to resist the infection much more successfully. In this patient, however, the temperature descended so quickly following the opening and cleansing of the wound and also the disappearance of the rigidity of the neck and the positive Kernig tests occurred so quickly, together with a lowering of the cell count of the cerebrospinal fluid, that naturally no cranial operation was considered necessary.

If the cerebrospinal fluid at lumbar puncture in this patient had been cloudy and yet no bacteria present, then the cranial operations would have been advisable as giving the patient a definite chance of recovery, but if bacteria had been found in the cerebrospinal fluid, then the condition would not have been a localized meningitis as in the former case, but a diffuse one, and therefore beyond the realm of surgery from the standpoint of an operation being of any real benefit to the patient.

If the dura underlying these linear fractures of the frontal bone had been torn in this patient, it is very probable that the infection of the scalp would have extended intracranially and therefore a purulent meningitis would undoubtedly have occurred. The importance of cleansing all scalp wounds carefully, together with a shaving of the surrounding scalp, is self-evident; if depressed fractures of the vault are excluded, this case-history is another illustration of the comparative unimportance of the fracture of the skull in these patients having cranial injuries, as compared with the possibility of severe intracranial lesions. This patient was able to walk to the hospital himself and yet there were present 3 linear fractures of the skull; this is not unusual in children and merely indicates the necessity of examining all of these patients most carefully for fear that the cranial condition is a more severe one than is indicated from a superficial examination alone, and therefore the proper treatment can be instituted early.

B. *Acute brain injuries associated with a mild increase of the intracranial pressure. Repeated lumbar punctures and drainage. Excellent recovery.*

Case 144.—Acute severe brain injury associated with a subdural hemorrhage and with a mild increase of the intracranial pressure; several Jacksonian convulsive seizures. Repeated lumbar punctures and drainage. Excellent recovery.

No. 84.—James. Five years. Black. U. S.

et at no time did the signs of a high intracranial pressure right hemiparesis gradually disappeared within a week and e an uneventful recovery.

ion at discharge (30 days after injury).—Temperature, 84; respiration, 26; blood-pressure, 112. No complaints other ess over the left half of vault; patient says: "I am all right." ver left parietal area has been absorbed. No weakness of right leg can be elicited by special tests. Impairment of hearing of e conduction is greater than air conduction; otoscopic examina- a small laceration in the posterior half of the left tympanic Pupils equal and react normally. Reflexes—patellar active, bly more than left; no ankle clonus and no Babinski, but there ar flexion to be obtained on right foot; abdominal reflexes both right possibly less active than left. Fundi—retinal veins slightly details of both optic disks clear. X-ray report (Doctor A. J. —"no fracture of the skull observed."

nation (January 8, 1917—29 months after injury).—No com- Goes to school daily; is not considered a "nervous" child; has convulsion since the day of the injury. Hearing of left ear normal; examination negative. Reflexes rather active but otherwise nega- ndi negative.

Examination (May 24, 1919—69 months after injury).—No com- Possibly more irritable than the other children; does well in school. possibly increased but otherwise negative. Fundi negative.

arks.—It is fortunate that such a good result was obtained in this and yet a longer period of time must elapse before it can be stated y degree of certainty that a normal patient has been obtained; the danger of convulsive seizures later in life and an emotional instability, nd without a mental retardation, must always be considered. If such ent should be examined now in the acute stage following the injury, I hat a left subtemporal decompression and drainage would offer the nt not only a greater chance of recovery of life but also of future nor- y, and it is my opinion that even though an apparently good result was ned in this patient by the expectant palliative treatment and the spinal age by repeated lumbar punctures, yet the patient underwent a much ter risk—both in the immediate results and in the remote effects of the ry—than if an early left subtemporal decompression and drainage had performed as soon as the initial shock had subsided—that is, in this ent within 8 hours after the injury. The localized Jacksonian convul- ns of the right arm and right leg and then the subsequent weakness of right side of the body which persisted for several days and associated th bloody cerebrospinal fluid—these signs indicated a localized lesion of e left cerebral cortex—most probably a supracortical hemorrhage or merely localized cortical edema but of sufficient amount to cause this impairment; the repeated lumbar punctures and drainage were sufficient to relieve the general intracranial pressure and thereby overcome the local effects of the supracortical hemorrhage or cortical edema, but whether this method of drainage was sufficient to remove the lesion, so that there will be no remote signs of its presence in the form of cortical adhesions, a partial blockage of the cerebrospinal fluid so that a mild condition of external hydrocephalus will result in the form of a mild degree of "wet," edematous brain —these considerations can only be accurately judged by the later condition of this patient as revealed by future examinations. In the present development of the operative technic of cranial operations, and particularaly of subtemporal decompressions and drainage, the risk is such a slight one that in all doubtful cases, such as this one, the operation should now be advised rather than the patient be permitted to risk such serious future complications.

Case 145.—Acute severe brain injury not associated with a fracture of the skull nor with a marked increase of the intracranial pressure; localized Jacksonian convulsions for 8 hours after the injury. No operation; repeated lumbar punctures and drainage. Excellent recovery.

No. 88.—William. Eight years. White. School. U. S.

Admitted August 21, 1914. Polyclinic Hospital. Referred by Doctor W. S. Bainbridge.

Discharged (at own risk) August 26, 1914—5 days after injury.

Family history negative.

Personal History.—Four years ago, patient fell from a one-story window, striking his head upon the ground; loss of consciousness for one hour; no bleeding from nose, mouth or ears; remained at home in bed for ten days and since then has been perfectly well; no convulsive seizures at any time.

Present Illness.—While playing baseball in the street, patient was knocked down by an automobile; unconscious for several minutes; brought to the hospital in the ambulance.

Examination upon admission (50 minutes after injury).—Temperature, 99°; pulse, 120; respiration, 24; blood-pressure, 114. Semiconscious; in practically no shock; answers questions when aroused and he then complains of severe headache. Contusion and hematoma of the right parietal area; definite tenderness. No bleeding from the nose, mouth or ears; no orbital or mastoid ecchymoses. No paralyses elicited but the left arm and left leg are not so relaxed as the right arm and right leg. (At this stage of the examination, the left leg and the left arm of the patient began to twitch and to shake spasmodically—there being no tonic spasm but frequently repeated clonic contractions; left side of face not involved; apparently no loss of consciousness. This localized convulsive seizure continued for over five minutes, did not become general and gradually ceased—the contractions of the left leg being the last to disappear; no biting of the tongue nor involuntary micturition or defecation occurred.) Pupils: before the convulsion, the pupils were equally enlarged but with normal reaction to light, whereas after the convulsive seizure, they became equally contracted and it was difficult to elicit any reaction to light. (Child had be very restless—continually turning and twisting in bed and requiri straint.) Reflexes: patellar exaggerated, left more than right; exha left ankle clonus and left Babinski; abdominal reflexes—left less activ right. Fundi negative. Lumbar puncture—clear cerebrospinal fluid slightly increased pressure (approximately 10 mm.); 12 c.c. slowly r as a therapeutic measure of drainage to lessen the acute cerebral (excess cerebrospinal fluid "water-logging" the cerebral tissues entirely or only in areas). X-ray (Doctor A. J. Quimby)—"no fracture of the skull observed."

Treatment.—Expectant palliative method assisted by repeated daily lumbar punctures for 3 days in order to drain any excess cerebrospinal fluid or cerebral edema. Forty minutes after the first convulsion, a second similar convulsive seizure occurred and again lasted almost 5 minutes; no loss of consciousness; the pupils remained equally contracted while the reflexes persisted in being exaggerated upon the left side and associated with a left Babinski; the retinal veins, however, became slightly enlarged, but no edematous obscuration of the details of either optic disk appeared. Two hours after admission, a second lumbar puncture was performed and 16 c.c. of clear cerebrospinal fluid under a pressure of approximately 10 mm. were carefully removed, until the pressure was only approximately 8 mm. A third convulsive seizure of the left leg and of the left arm occurred 5 hours after admission, but it was much less severe than the preceding ones and continued for only one-half minute; the neurological examination remained practically the same as before, while a third lumbar puncture removed 14 c.c. of clear cerebrospinal fluid under a slightly increased pressure of approximately 9 mm., and at the end of the drainage the pressure was approximately 7 mm. No further convulsive seizures occurred; the left arm and left leg were possibly slightly weaker than the right arm and right leg during the preceding examinations, but the difference was so slight that it could not be ascertained with certainty—the patient being right-handed and naturally stronger on the right side.

Examination (29 hours after admission).—Temperature, 99.4°; pulse, 94; respiration, 22; blood-pressure, 116. Rather drowsy but he is much brighter and answers questions easily and clearly. Hematoma over the right parietal bone remains boggy and not tense. No impairment of hearing; otoscopic examination negative. No weakness of the extremities nor can any sensory impairment be ascertained. Pupils rather contracted equally but react to light normally. Reflexes: patellar—left more active than right; no ankle clonus; left Babinski; abdominal reflexes—left possibly less active than right. Fundi—retinal veins slightly enlarged; no edematous blurring of the details of either optic disk. Lumbar puncture—clear cerebrospinal fluid under approximately 9 mm. pressure; 12 c.c. carefully withdrawn until the pressure was approximately 7 mm.

Treatment.—Expectant palliative continued. The improvement of the general condition of the patient rapidly progressed; he complained, however, of rather severe headache, although it was greatly lessened for about 6 hours following each lumbar puncture. Upon the following day, the fifth and last lumbar puncture was performed and 12 c.c. of cerebrospinal fluid under a pressure of approximately 9 mm. were carefully removed—the pressure at the end of the drainage being approximately 7 mm. The patient now felt so much better that he desired to get out of bed and to go home the following day; it was with difficulty that he could be kept in the hospital and he finally persuaded his parents to insist upon his discharge on the fifth remote signs of its presence in the form of cortical adhesions, a partial blockage of the cerebrospinal fluid so that a mild condition of external hydrocephalus will result in the form of a mild degree of "wet," edematous brain —these considerations can only be accurately judged by the later condition of this patient as revealed by future examinations. In the present development of the operative technic of cranial operations, and particularaly of subtemporal decompressions and drainage, the risk is such a slight one that in all doubtful cases, such as this one, the operation should now be advised rather than the patient be permitted to risk such serious future complications.

CASE 145.—Acute severe brain injury not associated with a fracture of the skull nor with a marked increase of the intracranial pressure; localized Jacksonian convulsions for 8 hours after the injury. No operation; repeated lumbar punctures and drainage. Excellent recovery.

No. 88.—William. Eight years. White. School. U. S.

Admitted August 21, 1914. Polyclinic Hospital. Referred by Doctor W. S. Bainbridge.

Discharged (at own risk) August 26, 1914—5 days after injury.

Family history negative.

Personal History.—Four years ago, patient fell from a one-story window, striking his head upon the ground; loss of consciousness for one hour; no bleeding from nose, mouth or ears; remained at home in bed for ten days and since then has been perfectly well; no convulsive seizures at any time.

Present Illness.—While playing baseball in the street, patient was knocked down by an automobile; unconscious for several minutes; brought to the hospital in the ambulance.

Examination upon admission (50 minutes after injury).—Temperature, 99°; pulse, 120; respiration, 24; blood-pressure, 114. Semiconscious; in practically no shock; answers questions when aroused and he then complains of severe headache. Contusion and hematoma of the right parietal area; definite tenderness. No bleeding from the nose, mouth or ears; no orbital or mastoid ecchymoses. No paralyses elicited but the left arm and left leg are not so relaxed as the right arm and right leg. (At this stage of the examination, the left leg and the left arm of the patient began to twitch and to shake spasmodically—there being no tonic spasm but frequently repeated clonic contractions; left side of face not involved; apparently no loss of consciousness. This localized convulsive seizure continued for over five minutes, did not become general and gradually ceased—the contractions of the left leg being the last to disappear; no biting of the tongue nor involuntary micturition or defecation occurred.) Pupils: before the convulsion, the pupils were equally enlarged but with normal reaction to light, whereas after the convulsive seizure, they became equally contracted and it was difficult to elicit any reaction to light. (Child had be
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as a therapeutic measure of drainage to lessen the acute cerebral

(excess cerebrospinal fluid "water-logging" the cerebral tissues entirely or only in areas). X-ray (Doctor A. J. Quimby)—"no fracture of the skull observed."

Treatment.—Expectant palliative method assisted by repeated daily lumbar punctures for 3 days in order to drain any excess cerebrospinal fluid or cerebral edema. Forty minutes after the first convulsion, a second similar convulsive seizure occurred and again lasted almost 5 minutes; no loss of consciousness; the pupils remained equally contracted while the reflexes persisted in being exaggerated upon the left side and associated with a left Babinski; the retinal veins, however, became slightly enlarged, but no edematous obscuration of the details of either optic disk appeared. Two hours after admission, a second lumbar puncture was performed and 16 c.c. of clear cerebrospinal fluid under a pressure of approximately 10 mm. were carefully removed, until the pressure was only approximately 8 mm. A third convulsive seizure of the left leg and of the left arm occurred 5 hours after admission, but it was much less severe than the preceding ones and continued for only one-half minute; the neurological examination remained practically the same as before, while a third lumbar puncture removed 14 c.c. of clear cerebrospinal fluid under a slightly increased pressure of approximately 9 mm., and at the end of the drainage the pressure was approximately 7 mm. No further convulsive seizures occurred; the left arm and left leg were possibly slightly weaker than the right arm and right leg during the preceding examinations, but the difference was so slight that it could not be ascertained with certainty—the patient being right-handed and naturally stronger on the right side.

Examination (29 hours after admission).—Temperature, 99.4°; pulse, 94; respiration, 22; blood-pressure, 116. Rather drowsy but he is much brighter and answers questions easily and clearly. Hematoma over the right parietal bone remains boggy and not tense. No impairment of hearing; otoscopic examination negative. No weakness of the extremities nor can any sensory impairment be ascertained. Pupils rather contracted equally but react to light normally. Reflexes: patellar—left more active than right; no ankle clonus; left Babinski; abdominal reflexes—left possibly less active than right. Fundi—retinal veins slightly enlarged; no edematous blurring of the details of either optic disk. Lumbar puncture—clear cerebrospinal fluid under approximately 9 mm. pressure; 12 c.c. carefully withdrawn until the pressure was approximately 7 mm.

Treatment.—Expectant palliative continued. The improvement of the general condition of the patient rapidly progressed; he complained, however, of rather severe headache, although it was greatly lessened for about 6 hours following each lumbar puncture. Upon the following day, the fifth and last lumbar puncture was performed and 12 c.c. of cerebrospinal fluid under a pressure of approximately 9 mm. were carefully removed—the pressure at the end of the drainage being approximately 7 mm. The patient now felt so much better that he desired to get out of bed and to go home the following day; it was with difficulty that he could be kept in the hospital and he finally persuaded his parents to insist upon his discharge on the fifth day after the injury; they accordingly signed the hospital release of all responsibility—"at own risk."

Examination at discharge (114 hours after injury).—Temperature, 98.8°; pulse, 88; respiration, 22; blood-pressure, 114. Perfectly conscious but rather irritable; upon questioning, patient admits he has a dull headache. Right parietal area slightly contused and boggy; no orbital ecchymoses. No weakness of the extremities elicited by the special tests. Pupils of normal size and reaction. Reflexes—equally active but otherwise negative; no Babinski. Fundi—retinal veins possibly slightly enlarged but otherwise negative.

Treatment.—Parents advised to keep the patient at home and in bed, if possible, and that he should lead a very quiet and inactive life for a period of 3 months at least; not to play in the hot sun; a vegetable diet; daily catharsis.

Examination (April 20, 1917—32 months after injury).—No complaints; attends school daily and "could not be better"; no headache nor convulsive seizure since his discharge from the hospital. Reflexes present and equal. Fundi negative.

Last Report (June 6, 1919—58 months after injury).—Letter from father states: "William is a well boy in every way; at times he becomes irritable but not more than the other children. No headache or fits. His teacher's reports are about the average."

Remarks.—The clinical history of this patient would indicate that a localized cerebral edema of the right motor cortex had occurred and of such mild degree that its irritative presence had produced the Jacksonian convulsive seizures of the left arm and left leg, and yet the cortex itself was not so water-logged or compressed as to produce a definite paralysis of the left arm and the left leg; the contraction of the pupils would confirm the irritative effect of the cortical lesion—the initial pupillary enlargement being undoubtedly due to the mild condition of the shock following the cranial injury. The repeated lumbar punctures and drainage of much clear cerebrospinal fluid undoubtedly facilitated the recovery of this patient, although even without the lumbar punctures it is probable that this patient would have made an excellent recovery under the expectant palliative treatment alone—merely a longer time being required for the convalescence; it is possible, however, that the cranial operation of right subtemporal decompression and drainage would have been indicated, and therefore it is considered that the spinal drainage formed an essential factor in the treatment of this patient and similarly selected patients, in whom the intracranial pressure is not so markedly increased that this method of spinal drainage would be a definite risk and danger to the patient.

The presence of the contusion of the scalp and the hematoma overlying the right parietal area would tend to point to the direct cranial injury as being an important factor in causing the localized cerebral edema directly beneath this part of the vault; the bilateral contraction of the pupils would indicate that the cortex of both hemispheres was equally irritated by the cortical edema, and therefore it is probable that the right motor cortex was only more so and to the extent of causing the localized convulsive seizures of the left arm and the left leg. It is possible for small supracortical subarachnoid hemorrhages to occur in these patients having injuries to the vault directly over the cerebral cortex and even numerous punctate hemorrhages within the cortex, but this complication is rather rare; in many patients, the cerebrospinal fluid at lumbar puncture will show the presence of blood and it is frequently advisable in these latter patients to perform the operation of cranial decompression and drainage, when the intracranial pressure is definitely increased—for fear of future complications and especially of convulsive seizures.

It is essential that patients of this character should receive most careful treatment in that all vigorous physical and mental work should be avoided for a period of months and that their lives should be regulated by careful hygienic rules—the avoidance of alcohol, meats, meat-soups, tea and coffee.

CASE 146.—Acute severe brain injury associated with a subdural hemorrhage and with a mild increase of the intracranial pressure; motor aphasia. Repeated lumbar punctures and drainage. Excellent recovery.

No. 704.—George. Eleven years. White. School. U. S.

Admitted October 17, 1916. Polyclinic Hospital.

Discharged October 31, 1916—14 days after injury.

Family history negative; both parents and grandparents were right-handed.

Personal History.—Negative. Patient is right-handed.

Present Illness.—While engaging in a street fight, patient was struck over the left side of his head with a small piece of lead pipe; apparently no complete loss of consciousness—merely stunned but he was unable to talk and gradually became stuporous. Patient was able to walk to the hospital supported by an older boy.

Examination upon admission (1 hour after injury).—Temperature, 99°; pulse, 74; respiration, 22; blood-pressure, 114. Rather stuporous and drowsy; unable to answer questions—merely shakes head for yes or no, but he is able to write the following: "I have a bad headache"; "My right hand feels like pins and needles"; "Everything is moving about me." No definite weakness of right side of the body nor any impairment of sensation could be elicited by special tests; no astereognosis nor apraxia. Small hematoma just below the left parietal crest; not particularly tender. No bleeding from the nose, mouth or ears; no orbital nor mastoid ecchymoses. Pupils equal and of normal reaction to light. Reflexes—patellar exaggerated, right more than left; exhaustible right ankle clonus and suggestive bilateral Babinski; abdominal reflexes—right absent. Fundi: retinal veins enlarged—left possibly more than right; nasal margin of left optic disk obscured by edema. Lumbar puncture—blood-tinged cerebrospinal fluid under a slightly increased pressure (approximately 10 mm.); 15 c.c. carefully removed.

Treatment.—Expectant palliative; for fear that a hemorrhage was occurring over the left cerebral cortex, the patient was repeatedly examined so that this serious complication could be recognized as early as possible and a left subtemporal decompression advised during the early stage, and thus the damaging effects of such local compression and the resulting general effects upon the medulla be anticipated and therefore avoided. The condition of the patient, however, remained practically the same, except that the ophthalmoscope, 6 hours after admission, revealed an edematous blurring of the nasal margins of both optic disks and a slight obscuration of a nasal half of the left optic disk—signs of an increasing intracranial pressure; a second lumbar puncture and drainage was now performed and 16 c.c. of blood-tinged cerebrospinal fluid were removed—the pressure at the beginning being approximately 12 mm. and at the end of the puncture only 9 mm. An almost immediate improvement appeared in that the patient became brighter and more alert, was able to say several words—among them being "I feel much better." A third lumbar puncture was performed upon the following day and 14 c.c. of straw-colored cerebrospinal fluid were removed—the pressure at the beginning being only approximately 10 mm. and at the end of the puncture approximately 8 mm.; the general and local condition of the patient continued to improve—speech was more smooth and of larger vocabulary, the headache less severe and the numbness and tingling of the right side of the body disappeared. A fourth lumbar puncture, however, was performed upon the following day and 15 c.c. of slightly straw-colored fluid were withdrawn—the pressure at the beginning being only approximately 9 mm. and at the end of the puncture being only 7 mm. From this time on, the patient made an uneventful recovery.

Examination at discharge (14 days after admission).—Temperature, 98.6°; pulse, 82; respiration, 24; blood-pressure, 116. Apparently normal mentally and emotionally; a very slight difficulty in speech but only to be elicited by test phrases; no sensory aphasia. No weakness of the right side of the body nor impairment of sensation. Hearing negative; otoscopic examination negative. Pupils negative. Reflexes: patellar active, right possibly more than left; no ankle clonus nor Babinski; abdominal reflexes —right possibly less active than left. Fundi—retinal veins slightly enlarged; lower portion of nasal margins of left optic disk indistinct from edema. X-ray report (Doctor William H. Stewart)—"no fracture of the skull is shown."

Examination (January 10, 1918—15 months after injury).—No complaints referable to the head injury. No impairment of speech can be obtained by special test phrases and patient stands well in his school classes. Reflexes active but otherwise negative. Fundi negative.

Last Report (April 20, 1919—30 months after injury).—Father writes: "Except for an attack of typhoid fever, George has been well and has no complaints. Will smoking hurt him?"

Remarks.—If this patient had shown a higher intracranial pressure, it would then have been advisable to have performed an early left subtemporal decompression and drainage rather than to have run the risk of a permanent damage to the underlying cerebral cortex; also if Jacksonian convulsive seizures had occurred, then too, it would have been dangerous to have delayed the cranial operation and drainage. The patient being repeatedly examined and under close observation and the intracranial pressure not being high, it was considered a rational method of treatment to perform repeated lumbar punctures and drainage and then, if the condition of the patient did become worse or if complications appeared, then the cranial operation of left subtemporal decompression and drainage could be advised. The excellent result obtained in this patient would tend to justify this method of treatment in certain selected patients and especially in children.

It is very rare for cases of pure motor aphasia to occur and for such an excellent recovery to result; usually the motor aphasia is associated with some degree of sensory aphasia or even with the condition, more or less complete, of astereognosis and also of apraxia. This condition of pure motor aphasia possibly occurs most frequently in these traumatic patients due to a localized supracortical hemorrhage or even to a localized cortical edema of mild degree. The parents and grandparents all being right-handed and the patient himself also being right-handed, would place the motor speech area in the left cerebral cortex. The absence of an overlying fracture of the skull at the site of the area of contact of the lead pipe tends to confirm again the statement that the fracture of the skull is possibly the most unimportant factor in brain injuries.

CASE 147.—Acute severe brain injury associated with extensive linear fractures of the vault but with only a mild increase of the intracranial pressure. No operation; repeated lumbar punctures and drainage. Excellent recovery.

No. 826.—Peter. Six years. White. School. U. S.

Admitted April 15, 1917. Polyclinic Hospital.

Discharged May 3, 1917—18 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While playing upon the roof of a shed, patient fell to the ground—a distance of 30 feet, striking upon the left side of head; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (50 minutes after injury).—Temperature, 98°; pulse, 130; respiration, 32; blood-pressure, 96. Semiconscious; in profound shock. Extensive bruise over left forehead with a bogginess in the left temporo-parietal area. Small amount of clotted blood in left nostril; no bleeding from mouth or ears; no mastoid ecchymoses. Diffuse left conjunctival ecchymosis; left orbital tissues so swollen and ecchymosed that patient cannot open left eye. No paralyses ascertained. Pupils enlarged and react to light sluggishly. Reflexes all abolished. Fundi negative.

Treatment.—On account of the severity of the shock, only a superficial examination was made at this time. Vigorous measures immediately instituted in the hope that the shock could be survived: rectal enemata of hot black coffee, heated blankets and several hot water bags; absolute quiet. Within 3 hours the general condition had improved, and at the end of 8 hours the severe condition of shock had almost disappeared.

Examination (12 hours after admission).—Temperature, 99°; pulse, 96; respiration, 26; blood-pressure, 110. Stuporous but can be aroused to answer questions in a confused manner. Boggy ecchymosis of left orbit and left side of head—a "doughy" feeling as of fluid in the subcutaneous tissues of the scalp. Marked area of tenderness over the left half of the frontal bone. No paralysis or impairment of sensation. Pupils—left possibly larger than right (most probably due to the local orbital condition); reaction to light normal. Reflexes—patellar present and equal; no ankle clonus but suggestive right Babinski; abdominal reflexes absent. Fundi—retinal veins enlarged; mild blurring of nasal margins of both optic disks. Lumbar puncture—bloody cerebrospinal fluid under a slightly increased pressure (11 mm.); 15 c.c. removed slowly and carefully. X-ray (Doctor G. W. Welton)—"a line of fracture extends through the left vertical plate of the frontal bone backward and forks—one upward and terminating near the coronal suture, while the other passes obliquely downward into the left parietal bone; another line of fracture extends forward through the right half of the frontal bone" (Fig. 165).

Treatment.—In the hope that the expectant palliative treatment aided by repeated lumbar punctures to drain the hemorrhage would be sufficient, this patient was actively treated in the routine manner and a steady daily improvement occurred. Repeated daily lumbar punctures were performed upon five consecutive days and at each time 15 c.c. of bloody cerebrospinal fluid were carefully and slowly removed, so that on the fifth day the pressure of the cerebrospinal fluid was only 9 mm. and straw-colored, whereas on the second day it had been 12 mm. and bloody. The boggy ecchymosis and edema of the left side of the scalp persisted for 10 days and then gradually subsided—apparently an excellent means of drainage of intracranial hemorrhage and cerebrospinal fluid through the lines of fracture.

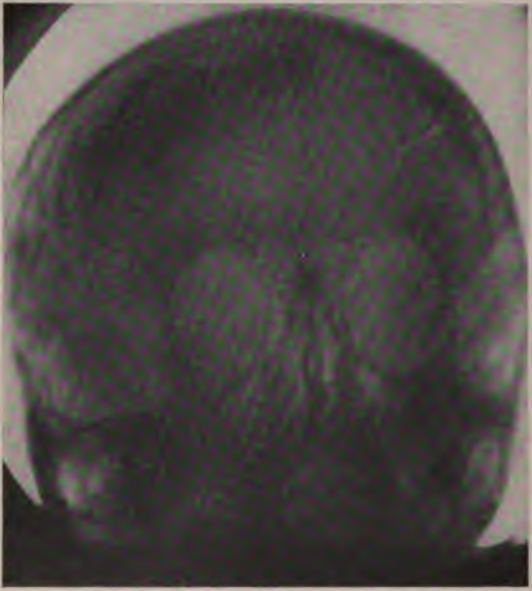

FIG. 165.—Multiple linear fractures of the anterior portion of the vault, in a patient having a mild increase of the intracranial pressure; spinal drainage by means of repeated lumbar punctures. Excellent recovery.

Examination at discharge (18 days after injury).—Temperature, 98.6°; pulse, 82; respiration, 24; blood-pressure, 114. No complaints except for general soreness over the left half of the head. Small amount of left subconjunctival hemorrhage persists. Pupils equal and react normally. Reflexes negative. Fundi—retinal veins slightly enlarged; margins of optic disks clear and distinct.

Treatment.—Parents advised that the child should not be allowed to play vigorously or become unusually excited for a period of three months; the avoidance of meat, meat-soups, tea and coffee; the importance of a daily movement of the bowels was emphasized.

Examination (February 20, 1918—10 months after injury).—No complaints, except his school-teacher states that child "is not as bright as formerly." Reflexes negative. Fundi negative.

Last Examination (May 6, 1919—25 months after injury).—No complaints; father states that "Peter is as well as ever and his school reports are good." Pupils equal and react normally. Reflexes negative. Fundi negative.

Remarks.—The extensive linear fractures of the left vault undoubtedly provided a means for the escape of intracranial hemorrhage and excess cerebrospinal fluid, and thus aided in the lowering of the intracranial pressure so that the operation of decompression and drainage could be avoided; whether this means in itself would have sufficed in this patient unless it had been aided by the repeated lumbar punctures and spinal drainage cannot be asserted with accuracy, but in some patients these linear fractures of the vault making possible the escape of intracranial hemorrhage and edema into the subcutaneous tissues of the scalp to form hematomata and diffuse boggy ecchymoses, are of definite therapeutic value. The danger of infection of the hematomata in these patients is a definite one, if the overlying scalp is very tense for a period of days or if the scalp itself is badly bruised and thus its resistance to infection lowered; otherwise, the risk is slight.

This case illustrates the comparative unimportance of the fracture of the skull, as a diagnostic means of the intracranial lesion and the necessity of a cranial operation or not; in fact, in this particular patient the fractures of the vault facilitated the treatment of the patient and aided in making unnecessary a cranial operation for decompression and drainage.

The therapeutic value of repeated lumbar punctures is well illustrated, not only as a means of estimating accurately the important intracranial status of the patient, but of the greatest importance in children and only less so in adults, as a valuable means of drainage of both subdural blood and excess cerebrospinal fluid. Naturally, if the increased intracranial pressure is high, then this method of spinal drainage must not be attempted for fear of inducing most serious medullary complications of direct compression, but in the milder patients in whom the intracranial pressure does not exceed 15 mm. as registered by the spinal mercurial manometer, then this method of spinal drainage can at least be used in the hope that it will be sufficient to prevent the necessity of the cranial operation of decompression and drainage; by careful estimation of the pressure of the cerebrospinal fluid at each puncture, it can be easily ascertained whether the intracranial pressure is gradually being lessened or not, and thus the patient is being afforded every chance of recovery at the least risk; if the spinal punctures are not sufficient in themselves to lower the increased intracranial pressure, then the operation of subtemporal decompression and drainage can be advised early and before the patient has reached the dangerous condition of severe medullary compression.

Case 148.—Acute severe brain injury associated with extensive comminuted linear fractures of vault and of the base of the skull, but with only a mildly increased intracranial pressure. No operation; repeated lumbar punctures and drainage. Excellent recovery.

No. 949.—George. Seven years. White. U. S.

Admitted February 13, 1918. Polyclinic Hospital.

Discharged February 28, 1918—15 days after injury.

Family history negative.

Personal history negative.

Present Illness.—While playing in the street, patient was struck by an automobile; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (20 minutes after injury).—Temperature, 98.6°; pulse, 90; respiration, 28; blood-pressure, 100. Unconscious; in mild shock. Multiple contusions and extensive hematomata of entire scalp—left half more than right. Profuse bleeding from left ear; left mastoid ecchymosis. Right orbital tissues ecchymosed but no subconjunctival hemorrhage. No paralysis elicited. Pupils rather small but react to light normally. Reflexes—patellar absent; no ankle clonus but left Oppenheim and Babinski reflex; abdominal reflexes could not be elicited. Fundi negative.

Treatment.—Owing to the presence of shock, a more thorough examination was not made at this time; immediate routine treatment of the shock was instituted and within four hours the general condition of the patient was much improved. Head carefully shaved and a mild (1–5000) bichloride dressing applied.

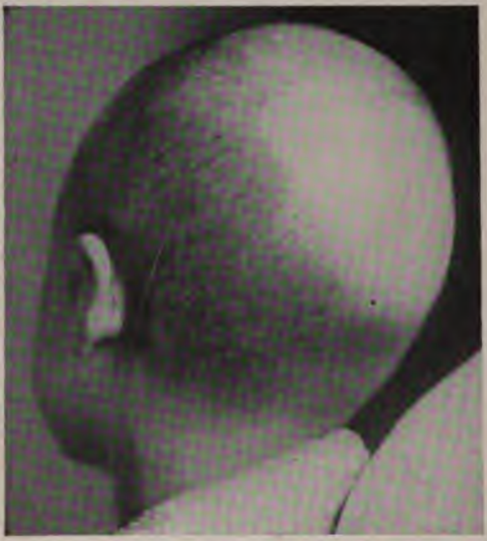

Fig. 166.—Boggy left mastoid ecchymosis in a patient who bled profusely through a torn left tympanic membrane. Excellent recovery with the expectant palliative treatment.

Examination (8 hours after admission).—Temperature, 99°; pulse, 88; respiration, 26; blood-pressure, 108. Semiconscious but cannot be aroused to answer questions. Entire scalp boggy and ecchymotic; not tense. Apparent crepitus can be elicited over the vertex. Discharge of blood from the left ear had ceased; otoscopic examination revealed a large laceration in the lower posterior quadrant of the left tympanic membrane. No paralysis ascertained. Left mastoid and right orbital ecchymoses (Figs. 166 and 167). Pupils of normal and equal size with normal reaction to light. Reflexes—patellar present, left possibly more than right; no ankle clonus but suggestive left Oppenheim and Babinski reflexes; abdominal reflexes depressed but apparently equal. Fundi—retinal veins enlarged; hazy blurring of the lower nasal margins of both optic disks. Lumbar puncture—blood-tinged cerebrospinal fluid under a slightly increased pressure (10 mm.); 15 c.c. carefully removed. X-ray (Doctor G. W. Welton)—"extensive linear fractures of the vault of the skull—more on the left side" (Fig. 168).

Examination (February 20, 1918—10 months after injury).—No complaints, except his school-teacher states that child "is not as bright as formerly." Reflexes negative. Fundi negative.

Last Examination (May 6, 1919—25 months after injury).—No complaints; father states that "Peter is as well as ever and his school reports are good." Pupils equal and react normally. Reflexes negative. Fundi negative.

Remarks.—The extensive linear fractures of the left vault undoubtedly provided a means for the escape of intracranial hemorrhage and excess cerebrospinal fluid, and thus aided in the lowering of the intracranial pressure so that the operation of decompression and drainage could be avoided; whether this means in itself would have sufficed in this patient unless it had been aided by the repeated lumbar punctures and spinal drainage cannot be asserted with accuracy, but in some patients these linear fractures of the vault making possible the escape of intracranial hemorrhage and edema into the subcutaneous tissues of the scalp to form hematomata and diffuse boggy ecchymoses, are of definite therapeutic value. The danger of infection of the hematomata in these patients is a definite one, if the overlying scalp is very tense for a period of days or if the scalp itself is badly bruised and thus its resistance to infection lowered; otherwise, the risk is slight.

This case illustrates the comparative unimportance of the fracture of the skull, as a diagnostic means of the intracranial lesion and the necessity of a cranial operation or not; in fact, in this particular patient the fractures of the vault facilitated the treatment of the patient and aided in making unnecessary a cranial operation for decompression and drainage.

The therapeutic value of repeated lumbar punctures is well illustrated, not only as a means of estimating accurately the important intracranial status of the patient, but of the greatest importance in children and only less so in adults, as a valuable means of drainage of both subdural blood and excess cerebrospinal fluid. Naturally, if the increased intracranial pressure is high, then this method of spinal drainage must not be attempted for fear of inducing most serious medullary complications of direct compression, but in the milder patients in whom the intracranial pressure does not exceed 15 mm. as registered by the spinal mercurial manometer, then this method of spinal drainage can at least be used in the hope that it will be sufficient to prevent the necessity of the cranial operation of decompression and drainage; by careful estimation of the pressure of the cerebrospinal fluid at each puncture, it can be easily ascertained whether the intracranial pressure is gradually being lessened or not, and thus the patient is being afforded every chance of recovery at the least risk; if the spinal punctures are not sufficient in themselves to lower the increased intracranial pressure, then the operation of subtemporal decompression and drainage can be advised early and before the patient has reached the dangerous condition of severe medullary compression.

Case 148.—Acute severe brain injury associated with extensive comminuted linear fractures of vault and of the base of the skull, but with only a mildly increased intracranial pressure. No operation; repeated lumbar punctures and drainage. Excellent recovery.

No. 949.—George. Seven years. White. U. S.

plaints; goes to school daily and no mental or emotional impairment has been observed by teacher. Hearing of left ear of normal acuity and the air conduction is now greater than the bone conduction; otoscopic examination—left tympanic membrane is negative. Reflexes negative. Fundi negative.

Remarks.—The linear fractures of the vault of this patient were possibly the most extensive I have ever seen; upon bimanual examination, distinct crepitus could be elicited—almost a crackling sensation upon gentle bimanual pressure. In this manner, such an excellent natural decompression had been formed and the escape and drainage of the intracranial hemorrhage and excess cerebrospinal fluid had been so profuse, that the increased intracranial pressure did not reach a height greater than 11 mm. and naturally, no cranial operation of decompression and drainage was advisable or at all necessary. The danger of an infective process extending through the scalp into the multiple hematomata and then the great risk of a purulent meningitis resulting, was less in this patient on account of the small amount of bruising of the scalp and the absence of a marked tenseness of the hematomata themselves; the careful shaving of the entire head and the application of a mild antiseptic wet dressing of bichloride (1–5000) assisted in lessening this danger of infection.

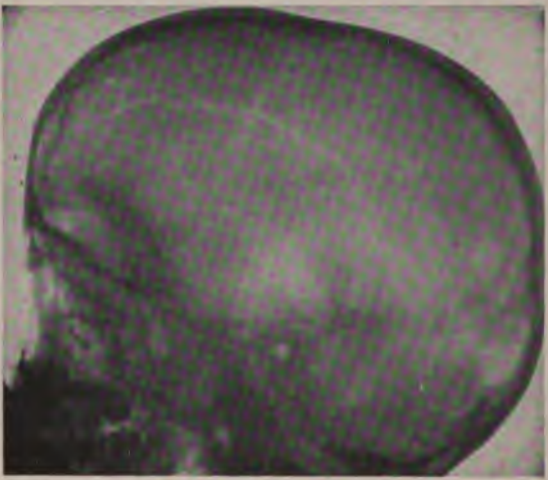

FIG. 168.—Extensive linear fractures of the anterior portion of the vault, in a patient having a mild increase of the intracranial pressure. Excellent recovery following the spinal drainage of repeated lumbar punctures and the expectant palliative treatment.

This patient is another illustration of the comparative unimportance of linear fractures of the skull in the diagnosis and treatment of brain injuries—unless considered as an aid in their treatment as in this patient. The presence of the left Oppenheim and Babinski reflexes for 2 days following the injury and then their gradual disappearance, was due most probably to a mild edematous condition of the right cerebral cortex—and not to a definite hemorrhagic clot formation, which would tend to prolong these signs of cortical and pyramidal tract involvement. Temporary edema of the cerebral cortex is a rather common occurrence in brain injuries in children and may be sufficient to cause localized epileptiform seizures, although in the presence of this latter complication, an immediate homolateral subtemporal decompression and drainage should be performed in order to lessen early this cortical irritation and thus avoid future complications.

The rapid improvement of the hearing of the left ear with the early healing of the laceration of the left tympanic membrane is most impressive

Expectant palliative; repeated daily lumbar punctures hope that they would facilitate the natural absorption of the racranial pressure, prevent it from ascending to a height a cranial operation and at the same time be a means of he small amount of free blood in the cerebrospinal fluid. The linear fractures, permitting the intracranial hemorrhage to escape the left ear and also into the subcutaneous tissues of the scalp and forming multiple hematomata with and without the mixture of rospinal fluid, were a fortunate means of lessening the increased intra- nial pressure and thus avoiding the necessity of a cranial operation decompression and drainage. Repeated daily lumbar punctures were performed upon four successive days and 15 c.c. of blood-tinged cerebrospinal fluid slowly and carefully removed. On the second day, the pressure was increased (11 mm.), whereas on the fourth day it was only 9 mm. The complaint of headache was relieved each time following the lumbar puncture and removal of the fluid.

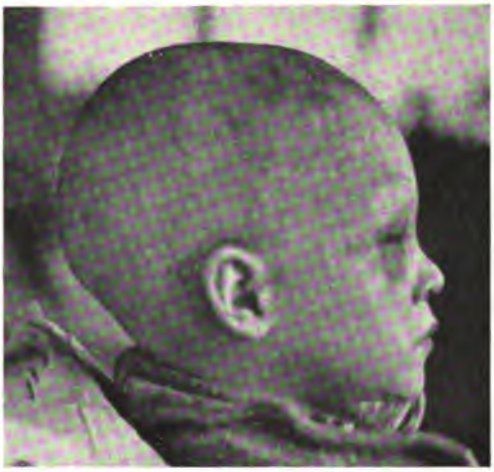

FIG. 167.—Right orbital and multiple scalp ecchymoses in a patient having multiple fractures of the vault and of the base. The right mastoid ecchymosis is indistinctly shown. Excellent recovery with the expectant palliative treatment.

Examination at discharge (15 days after injury).—Temperature, 98.6°; pulse, 82; respiration, 24; blood-pressure, 112. No complaints except a general soreness of the head, especially on the left side. Several ecchymotic areas throughout the scalp and over the left mastoid. No paralyses nor impairments of sensation. Hearing less in left ear and bone conduction was greater than air conduction. Pupils equal and react normally. Fundi—retinal veins slightly enlarged; details of optic disks clear and distinct.

Treatment.—Parents cautioned regarding the diet of the child and especially the avoidance of meat, meat-soups, tea and coffee; daily regulation of the bowels; the avoidance of much excitement and vigorous play and at least 10 hours' sleep each night—and better 12 hours; no school until the fall—7 months later.

Examination (August 22, 1918—6 months after injury).—No complaints; apparently "the same as before the injury." Palpation of left side of vault elicits slight tenderness. Hearing of left ear less acute than of right; otoscopic examination reveals a healed scar in the posterior lower quadrant of the left tympanic membrane; bone conduction equals air conduction. Reflexes—present and equal; normal plantar flexion and reflexes. Fundi negative.

Last Examination (May 10, 1919—15 months after injury).—No complaints; goes to school daily and no mental or emotional impairment has been observed by teacher. Hearing of left ear of normal acuity and the air conduction is now greater than the bone conduction; otoscopic examination—left tympanic membrane is negative. Reflexes negative. Fundi negative.

Remarks.—The linear fractures of the vault of this patient were possibly the most extensive I have ever seen; upon bimanual examination, distinct crepitus could be elicited—almost a crackling sensation upon gentle bimanual pressure. In this manner, such an excellent natural decompression had been formed and the escape and drainage of the intracranial hemorrhage and excess cerebrospinal fluid had been so profuse, that the increased intracranial pressure did not reach a height greater than 11 mm. and naturally, no cranial operation of decompression and drainage was advisable or at all necessary. The danger of an infective process extending through the scalp into the multiple hematomata and then the great risk of a purulent meningitis resulting, was less in this patient on account of the small amount of bruising of the scalp and the absence of a marked tenseness of the hematomata themselves; the careful shaving of the entire head and the application of a mild antiseptic wet dressing of bichloride (1–5000) assisted in lessening this danger of infection.

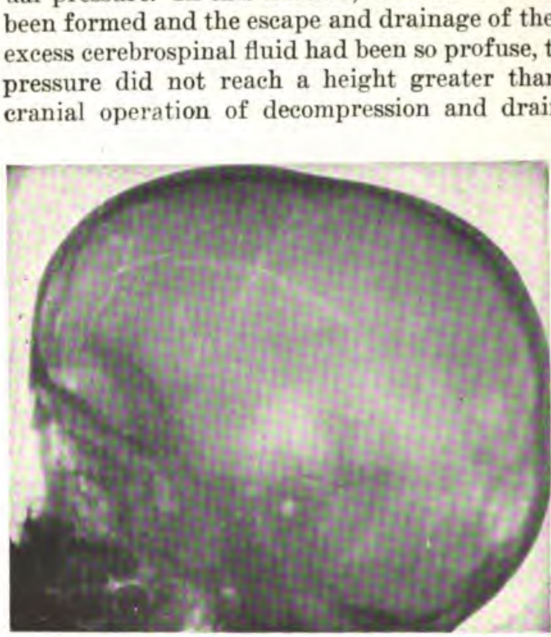
FIG. 168.—Extensive linear fractures of the anterior portion of the vault, in a patient having a mild increase of the intracranial pressure. Excellent recovery following the spinal drainage of repeated lumbar punctures and the expectant palliative treatment.

This patient is another illustration of the comparative unimportance of linear fractures of the skull in the diagnosis and treatment of brain injuries—unless considered as an aid in their treatment as in this patient. The presence of the left Oppenheim and Babinski reflexes for 2 days following the injury and then their gradual disappearance, was due most probably to a mild edematous condition of the right cerebral cortex—and not to a definite hemorrhagic clot formation, which would tend to prolong these signs of cortical and pyramidal tract involvement. Temporary edema of the cerebral cortex is a rather common occurrence in brain injuries in children an
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and is characteristic of these middle ear traumatic impairments, where the tympanic membrane is the chief lesion. This is the usual result of similar lesions in adults, but in them the return to normality is not so rapid. The danger of infection through the ear in these patients is practically nil, unless meddlesome procedures of irrigation and "cleaning out" of the auditory canal are attempted.

C. *Acute cranial injuries associated with a depressed fracture of the vault. Removal of the depressed bone. Excellent recovery.*

Case 149.—Acute depressed fracture of the vault associated with an extensive linear fracture; no increase of the intracranial pressure. Removal of the depressed bone. Excellent recovery.

No. 728.—John. Four years. White. Holland.

Admitted November 26, 1917. Polyclinic Hospital.

Operation November 28, 1917—2 days after injury. Removal of depressed area of bone.

Discharged December 12, 1917—14 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While playing upon a fire-escape, child fell one flight to the cement pavement below; apparently no loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (40 minutes after injury).—Temperature, 98.4°; pulse, 110; respiration, 28; blood-pressure, 102. Very restless and crying; mild degree of shock. Hematoma over postero-superior portion of the right parietal bone. No bleeding from nose, mouth or ears; orbital and right mastoid ecchymoses. No paralyses. Pupils slightly enlarged but react normally. Reflexes—present and equal; no Babinski. Fundi negative.

Treatment.—Expectant palliative.

Examination (30 hours after admission).—Temperature, 99°; pulse, 84; respiration, 24; blood-pressure, 112. Apparently well and wants to go home. Entire scalp ecchymotic—particularly boggy over the vertex and to the right of the midline. Bimanual examination is negative except for tenderness, especially over the right side of the vault. Both orbits and right mastoid area are ecchymosed. No paralyses nor impairments of sensation. Pupils equal and react normally. Reflexes—patellar active but equal; no ankle clonus nor Babinski; abdominal reflexes present and equal. Fundi—retinal veins possibly enlarged; no obscuration of the details of either optic disk. Lumbar puncture—clear cerebrospinal fluid under normal pressure (8 mm.). X-ray (Doctor G. W. Welton)—"shows a depressed fracture of the vault at the postero-superior angle of the right parietal bone; there is a wide fracture line extending obliquely downward and forward to the base" (Fig. 169).

Treatment.—An exploratory incision over the depressed area of the vault was advised for fear of the possible complication of infection and also of the future danger of convulsive seizures.

Operation (40 hours after admission).—Removal of depressed area of vault; small horseshoe-shaped incision of the edematous and hemorrhagic scalp, exposing a depressed area of both tables of the vault of about the size of a silver quarter. In order to remove the depressed area of bone, a trephine opening was made upon either side of the depression, the opening enlarged and the fragment of bone taken out; a sharp-pointed portion pressing against the dura and tearing it was carefully removed; the underlying cortex apparently not damaged. It was possible to see and to feel the wide linear fracture extending downward from this area as disclosed by the röntgenogram. No attempt was made to suture the dural opening (the size of one-quarter inch in length) and a small drain of rubber tissue was inserted into this opening. Brain itself was not under increased pressure. Usual closure of the scalp with two drains of rubber tissue inserted down to the dura. Duration, 30 minutes.

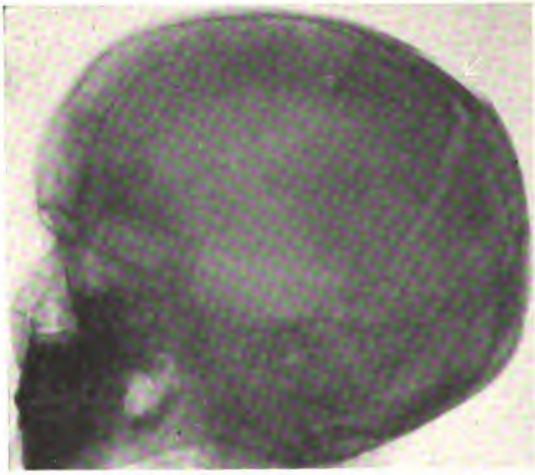


FIG. 169. —Huge linear fracture extending downward from a depressed fracture of the vault in a patient having no increase of the intracranial pressure. Excellent recovery following the expectant palliative treatment and a removal of the depressed area of bone.

Post-operative Notes.—Uneventful recovery and convalescence. Incision healed *per primam.*

Examination at discharge (14 days after operation).—Temperature, 98.6°; pulse, 82; respiration, 24; blood-pressure, 112. No complaints except general soreness of the head, especially the right side. Right orbit and right mastoid area are slightly ecchymotic. Hearing negative. Pupils negative. Reflexes negative. Fundi negative. The operative area is slightly depressed; normal pulsation; incision entirely healed so that no gauze dressing was necessary.

Examination (July 20, 1918—8 months after injury).—No complaints. Operative area depressed; slight pulsation visible. Reflexes negative. Fundi negative.

Last Examination (May 8, 1919—18 months after injury).—No complaints. Operative area depressed; slight pulsation visible. Reflexes negative. Fundi negative.

Remarks.—The importance of careful and routine X-ray pictures of all patients having cranial injuries is well emphasized in this case-histor
is possible that this depressed area of bone might have been overlool
this patient until months or even years after—when symptoms and
had developed, and then it would have been late for the best results
obtained. The ideal time for relieving the condition and for prev
untoward signs and complications is after the shock has been over
the removal of the depressed area of bone under strict asepsis also

the danger of meningitic complications from infection of the hematoma of the scalp and especially if the adjacent skin has been badly contused.

The absence of an increased intracranial pressure made it possible to remove the depressed bony fragment first and with safety without preceding it by the operation of subtemporal decompression, which should always first be performed if the intracranial pressure is definitely increased. The extensive fracture of the skull is interesting in view of the absence of unconsciousness and also the signs of severe shock. It merely emphasizes the comparative unimportance of the fracture of the skull, as an index of the severity of the brain injury—in fact, the presence of a fracture of the skull or not is of little importance—unless it is a depressed one of the vault, which should always be elevated or removed.

CASE 150.—Acute severe brain injury associated with extensive fractures of the vault and a depressed fracture of the right parietal bone; no signs of an intracranial lesion or of an increased intracranial pressure. Operation to remove the depression of the vault refused, but 5 months later consent was obtained and the operation was then performed. Excellent recovery.

No. 922.—John. Four years. White. Holland.

Admitted June 26, 1917. Polyclinic Hospital.

Discharged June 27, 1917—22 hours after admission. Operation refused.

Operation, November 28, 1917—5 months after injury. Removal of depressed area of vault.

Family history negative.

Personal history negative.

Present Illness.—While playing upon a fire-escape, child fell to the stone sidewalk below—a distance of 18 feet—striking upon the right side of head; merely stunned but no loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (40 minutes after injury).—Temperature, 98.6°; pulse, 108; respiration, 28; blood-pressure, 100. Rather stuporous and drowsy, but cries for his mother when aroused; in mild condition of shock. Contusion of scalp over right parietal area; careful palpation of this region reveals an apparent depression of the underlying bone and the sensation of a "crack" extending downward. No bleeding from nose, mouth or ears; no orbital or mastoid ecchymoses; otoscopic examination negative. No paralysis of the extremities elicited. Pupils equal and react to light normally. Reflexes—patellar present and equal; no ankle clonus nor Babinski; abdominal reflexes possibly depressed but equal. Fundi negative. Lumbar puncture—clear cerebrospinal fluid under normal pressure (8 mm.). X-ray report (Doctor G. W. Welton)—"bony depression of the lower posterior portion of the right parietal bone—almost 2 cm. in diameter; wide linear fracture—almost a defect—extends downward from this depressed area" (Fig. 170).

Treatment.—Expectant palliative method. Within one hour, the child reacted strongly so that the general condition was considered excellent; upon repeated examinations within the next 12 hours, the patient disclosed no neurological signs of an intracranial injury and there were no signs of an increased intracranial pressure. On account of the depression of the

Discharged October 10, 1918—15 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While playing upon a fire-escape, child fell headlong to the cement pavement—a distance of 25 feet—striking upon his right forehead; immediate loss of consciousness; patient was found by a policeman, lying upon the pavement, and was brought to the hospital.

Examination upon admission (about one hour after injury).—Temperature, 98°; pulse, 126; respiration, 30; blood-pressure, 102. Semiconscious; moderate degree of shock. Hematoma over the right half of frontal bone—the size of a lemon; not tense and palpation elicits crepitus and an apparent depression of the underlying bone. Clotted blood in both nostrils but no bleeding from the mouth or ears; right orbital ecchymosis but no mastoid discoloration. No paralyses elicited. Pupils—enlarged, equal and react to light sluggishly. Reflexes all abolished. Fundi negative. No further examination was made on account of the severity of shock.

Treatment.—Vigorous shock measures instituted; routine expectant palliative treatment. Child rapidly recovered from the shock so that in the morning, 12 hours later, the following examination was made:

Examination (12 hours after admission).—Temperature, 99°; pulse, 88; respiration, 24; blood-pressure, 114. Perfectly conscious; when asked how he was, he replied, "I feel fine." Right orbit closed by ecchymotic swelling, while the adjacent hematoma had become larger and more tense; only slightly tender. No paralyses or impairments of sensation. Pupils of normal size and reaction. Reflexes—patellar exaggerated but equal; no ankle clonus but double Babinski; abdominal reflexes absent. Fundi—retinal veins dilated: nasal halves and temporal margins of both disks blurred by edema. Lumbar puncture—blood-tinged cerebrospinal fluid under a mild pressure (11 mm.). X-ray (requested but unfortunately it was not taken until 3 days later, due to necessary repairs upon the X-ray machine) Doctor G. W. Welton—"large depressed fracture of the right half of the frontal bone; linear fracture extends vertically downward from this depressed area into the squamous portion of right temporal bone" (Fig. 171).

Treatment.—The signs of a definite increase of the intracranial pressure not becoming more marked, the patient was treated expectantly until it was definitely ascertained by the röntgenograms that a depressed fracture of the frontal bone was present. An immediate operation was advised; owing to the increased intracranial pressure, a right subtemporal decompression was performed first and then the removal of the depressed area of the vault followed.

Operations (4 days after admission).—*First.* Right subtemporal decompression: usual vertical incision, bone removed, and no complications: in the temporal muscle beneath the temporal fascia was found much free clotted blood, indicating a fracture of the underlying bone, which was found extending backward along the upper portion of the squamous bone; a small amount of extradural clotted blood removed. Dura tense and bluish; upon incising it, bloody cerebrospinal fluid spurted to a height of 2 inches, revealing a "wet," œdematous cortex but no cortical hemorrhages or lacerations make a small trephine opening at the anterior edge; the rongeurs were inserted, the opening enlarged and the depressed bony fragment was removed. It was found that a small tear of the underlying dura had been made by the pointed bony edge, but no other lesion was observed and the underlying cortex pulsated normally and under normal tension. A small piece of the pericranium was placed over the dural opening and the scalp sutured after 2 drains of rubber tissue had been inserted. Duration, 25 minutes.

Post-operative Notes.—An uneventful convalescence occurred, so that child was discharged on the eighth day after operation—the neurological examinations being negative as before the operation. A post-operative röntgenogram was now taken, "showing the bony defect due to the removal of the depressed area of bone; descending line of fracture very faint."

Last Examination (May 21, 1919—23 months after injury and 18 months after operation).—No complaints—"as if the bump had not happened"; went to primary class during the past year and his reports were good. Site of operation pulsates slightly; bony opening smaller from new bone formation about its periphery. Reflexes negative. Fundi negative.

Remarks.—The extensive fractures of the skull in this patient, as demonstrated by the röntgenogram and at operation, in the absence of any intracranial lesion of hemorrhage or cerebral edema, illustrate again the comparative unimportance of the fractures of the skull following cranial injuries in the diagnosis and prognosis of these patients; in the absence of the depression of the vault, no operation would have been indicated, and it is possible for this patient to have obtained an excellent ultimate result even without the removal of the depressed area of the vault, and yet it was too great a risk to permit this child to undergo for fear that future complications might occur; therefore, the removal of the depressed area of the vault was advised in the absence of all neurological signs and merely as a prophylactic measure.

The clear cerebrospinal fluid as obtained by lumbar puncture immediately following the cranial injury tended to exclude an intracranial hemorrhage and this is usually true, although it is possible for an extensive subdural hemorrhage to be present and yet the cerebrospinal fluid at lumbar puncture be clear; this is probably due to a blockage of the cerebrospinal fluid in the neighborhood of the foramen magnum.

It must be remembered in these patients that the risk of the simple elevation and removal of a depressed fracture of the vault is practically nil, and that only primary anesthesia is necessary in children and the more nervous of the patients, while the local anesthesia of novocaine is very satisfactory for the more stable patients.

Case 151.—Acute severe brain injury associated with a depressed fracture of the skull and with marked signs of an increased intracranial pressure; laceration of the right frontal lobe. Subtemporal decompression first and then a removal of the depressed area of bone. Excellent recovery.

No. 1024.—Peter. Seven years. White. School. U. S.

Admitted September 21, 1918. Polyclinic Hospital.

Operation September 25, 1918—4 days after admission. First, right subtemporal decompression; second, removal of depressed area of vault.

not been demonstrated, then naturally no operation would have been indicated since the intracranial pressure was not high enough in itself to warrant the operation of decompression and drainage, and the presence of the overlying hematoma made impossible an accurate diagnosis of the underlying depressed fracture of the vault; when satisfactory röntgenograms are not available, then an exploratory incision of the scalp should be performed for fear of future complications, both mentally and emotionally, and especially in regard to convulsive seizures. In this patient a subtemporal decompression was first performed for fear of producing still greater damage to the underlying cerebral cortex, while attempting to remove the bony depression—a frequent disaster when this precaution is not taken.

The absence of clinical signs of the cortical laceration other than the temporary ones of increased reflexes, and particularly the Babinski (which was bilateral), is a common observation in patients when the comparatively "silent areas" of the cerebral cortex are involved—and possibly most of all, the right frontal lobe in right-handed patients and then the right temporosphenoidal lobe. Naturally, sufficient time has not elapsed since the injury to estimate the effects of this intracranial injury upon the future mental and emotional development of the child or whether he will continue to remain as normal as before the injury; it is very doubtful and yet similar cases of years in duration have had that fortunate end-result. The danger, however, of emotional instability at least is very great indeed.

D. *Acute brain injuries associated with an increased intracranial pressure. Subtemporal decompression and drainage. Excellent recovery.*

CASE 152.—Acute severe brain injury associated with a fracture of the base of the skull and with high intracranial pressure due to cerebral edema. Right subtemporal decompression and drainage. Excellent recovery.

No. 046.—Mary. Four years. White. U. S.

Admitted May 23, 1913—3 hours after injury. Polyclinic Hospital. Referred by Doctor J. A. Bodine.

Operation (May 23, 1913—3 hours after admission). Right subtemporal decompression and drainage.

Discharged June 5, 1913—12 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While playing in the street, child was struck by an express wagon; no known loss of consciousness, but she became very drowsy within one hour and bled from the right ear; brought to the hospital in the ambulance.

Examination upon admission (3 hours after injury).—Temperature, 100.4°; pulse, 66; respiration, 16; blood-pressure, 120. Well-nourished white child. Semiconscious; no signs of shock. Bimanual examination of head is negative. Small amount of blood trickling from the right ear; no cerebrospinal fluid observed. Right mastoid ecchymosis. Pupils equal and of normal reaction. Reflexes—knee-jerks active, right possibly greater than left; tendency to a right Babinski; abdominal reflexes present, left possibly greater than right. Fundi—definite fulness of retinal vessels with slight edematous haziness over the nasal halves of both optic disks. Lumbar observed. Cortex tended to bulge, but owing to the escape of much cerebrospinal fluid, it receded at the end of the operation and pulsated slightly. Usual closure with 2 drains of rubber tissue inserted.

Second. Removal of the depressed bone: vertical incision of 3 inches over the hematoma and the site of the depressed area of the vault. Upon retracting the scalp, much clotted blood and macerated brain tissue welled out of cavity, exposing a triangular depressed area of bone of 2 inches in diameter and extending through the torn dura into the underlying cortex. This fragment of depressed bone was carefully removed with rongeurs, the dura sutured and a small drain of rubber tissue inserted subdurally into the laceration of the brain. Usual closure with 2 drains of rubber tissue inserted beneath the scalp. Duration, 80 minutes.

Post-operative Notes.—Uneventful operative recovery; much bloody and then straw-colored cerebrospinal fluid was drained; all drains removed on the second day and sutures on the sixth day post-operative.

Examination at discharge (13 days after operation).—Temperature, 98.6°; pulse, 80; respiration, 24; blood-pressure, 114. No complaints except for soreness about the operative areas. No impairment mentally or emotionally elicited; no paralyses or sensory disturbances. Pupils equal and react normally. Reflexes: patellar active but equal; no ankle clonus nor Babinski—possibly a tendency to a Babinski on the left foot; abdominal reflexes—left depressed. Fundi—retinal veins possibly enlarged; no obscuration of details of either optic disk.

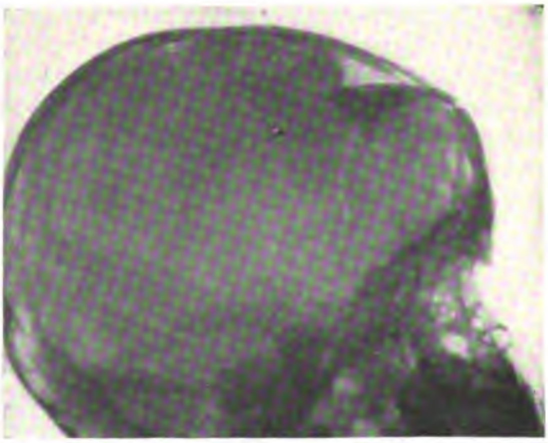


FIG. 171.—Huge depressed fracture and radiating wide linear fracture of right half of frontal bone in a patient having a high intracranial pressure; right subtemporal decompression first and then a removal of the depressed bone. Excellent recovery.

Treatment.—Parents cautioned to restrict the child's activities for a period of 3 months at least; no meat, meat-soup, tea or coffee; daily bowel movement.

Examination (June 10, 1919—4 months after injury).—No complaints; is going to school and "seems as well as ever." Operative sites slightly depressed but the pulsation is normal. Reflexes active but otherwise negative. Fundi negative—possibly slight enlargement of the retinal veins.

Last Examination (May 21, 1919—8 months after injury).—No complaints—"just like any other child." Operative sites more depressed than at preceding examination. Reflexes negative, except for a less active left abdominal reflex. Fundi negative.

Remarks.—The importance of an X-ray picture in all cranial injuries is well illustrated in this patient; if the depressed fracture of the vault had not been demonstrated, then naturally no operation would have been indicated since the intracranial pressure was not high enough in itself to warrant the operation of decompression and drainage, and the presence of the overlying hematoma made impossible an accurate diagnosis of the underlying depressed fracture of the vault; when satisfactory röntgenograms are not available, then an exploratory incision of the scalp should be performed for fear of future complications, both mentally and emotionally, and especially in regard to convulsive seizures. In this patient a subtemporal decompression was first performed for fear of producing still greater damage to the underlying cerebral cortex, while attempting to remove the bony depression —a frequent disaster when this precaution is not taken.

The absence of clinical signs of the cortical laceration other than the temporary ones of increased reflexes, and particularly the Babinski (which was bilateral), is a common observation in patients when the comparatively "silent areas" of the cerebral cortex are involved—and possibly most of all the right frontal lobe in right-handed patients and then the right temporosphenoidal lobe. Naturally, sufficient time has not elapsed since the injury to estimate the effects of this intracranial injury upon the future mental and emotional development of the child or whether he will continue to remain as normal as before the injury; it is very doubtful and yet similar cases of years in duration have had that fortunate end-result. The danger, however, of emotional instability at least is very great indeed.

D. *Acute brain injuries associated with an increased intracranial pressure. Subtemporal decompression and drainage. Excellent recovery.*

CASE 152.—Acute severe brain injury associated with a fracture of the base of the skull and with high intracranial pressure due to cerebral edema. Right subtemporal decompression and drainage. Excellent recovery.

No. 046.—Mary. Four years. White. U. S.

Admitted May 23, 1913—3 hours after injury. Polyclinic Hospital. Referred by Doctor J. A. Bodine.

Operation (May 23, 1913—3 hours after admission). Right subtemporal decompression and drainage.

Discharged June 5, 1913—12 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While playing in the street, child was struck by an express wagon; no known loss of consciousness, but she became very drowsy within one hour and bled from the right ear; brought to the hospital in the ambulance.

Examination upon admission (3 hours after injury).—Temperature, 100.4°; pulse, 66; respiration, 16; blood-pressure, 120. Well-nourished white child. Semiconscious; no signs of shock. Bimanual examination of negative. Small amount of blood trickling from the right ear; no spinal fluid observed. Right mastoid ecchymosis. Pupils equal normal reaction. Reflexes—knee-jerks active, right possibly great left; tendency to a right Babinski; abdominal reflexes present, left greater than right. Fundi—definite fulness of retinal vessels wi edematous haziness over the nasal halves of both optic disks.

puncture—cerebrospinal fluid slightly blood-tinged and under high pressure (approximately 21 mm.); only 4 c.c. removed for examination.

Treatment.—An immediate right subtemporal decompression and drainage advised to lower this high intracranial pressure for fear of acute medullary complications.

Operation (3 hours after admission).—Right subtemporal decompression (primary anesthesia): usual vertical incision and bone removed; no complications. Dura very tense, and upon incising it, the slightly blood-tinged cerebrospinal fluid spurted a distance of 9 inches—striking the operator in the left eye and passing even behind his head. The underlying cerebral cortex was very "wet" and edematous with its vessels markedly congested; no hemorrhage or cortical laceration visible. The bulging cortex became less tense as the excess cerebrospinal fluid escaped, so that at the end of the operation the pulsation of the brain was almost normal. Usual closure with one drain of rubber tissue inserted beneath the right temporo-sphenoidal lobe and emerging at the lower angle of the incision. Duration, 38 minutes.

Post-operative Notes.—Uneventful recovery and convalescence; the drain was removed on the second day upon the lessening of the drainage of clear cerebrospinal fluid. Child became perfectly conscious and improved rapidly.

Examination at discharge (12 days after operation).—Temperature, 98.8°; pulse, 84; respiration, 24; blood-pressure, 116. No complaints except the usual soreness at the site of operation; this bulged slightly but pulsated normally. Hearing of right ear less than left; bone conduction greater than air conduction in right ear; otoscopic examination discloses a tear in the upper posterior quadrant of the right tympanic membrane. Reflexes—active but otherwise negative. Fundi—retinal veins possibly enlarged but otherwise negative. X-ray report (Doctor A. J. Quimby)—"no fracture of the skull demonstrated."

Examination (June 4, 1914—13 months after injury).—No complaints. The operative site is being narrowed by new bone formation—slightly convex outward. Hearing of right ear almost equals that of left; bone conduction, however, equals air conduction. Reflexes negative. Fundi negative.

Examination (September 16, 1917—40 months after injury).—No complaints; goes to school daily and "no different from the other children," writes teacher. New bone formation has almost entirely covered the decompression opening except at its posterior middle portion; slight pulsation palpable there. Hearing of both ears equally acute; otoscopic examination of right ear negative, air conduction greater than bone conduction in both ears. Reflexes negative. Fundi negative.

Last Examination (May 4, 1919—72 months after injury).—No complaints referable to the head. A very small irregular bony opening at the site of the former operation persists; no pulsation visible or palpable. Hearing negative. Reflexes negative. Fundi negative.

Remarks.—The new bone formation which occurred in these patients before the periosteum of the bone was removed at the time of the operation is not in any way harmful, unless there persists a mild chronic cerebral edema producing an increase of the intracranial pressure; in these latter patients, it would then be essential for this pressure to be entirely relieved by the patent decompression opening and the decompressive effects in no degree lessened; if, however, the increased intracranial pressure has disappeared, then there is no need for the bony decompression opening to remain patent. For fear, however, that this intracranial pressure does not subside to normal, it is essential that the decompression opening remain a permanent one, and for this reason, the so-called periosteum covering this bone is always removed, and during the past three years there has not been a patient in whom the operative site has been covered by a membrane formation—at most merely a slight narrowing of the opening at the bony margins alone.

The excellent immediate and end-results of the patients of this character are most gratifying; a normal individual is obtained—emotionally, mentally and physically, with little or no risk of future complications such as epileptiform seizures, etc. The rapid recovery of normal hearing of the impaired ear is the usual history of these patients, and especially in children, with and without operation.

CASE 153.—Acute severe brain injury associated with a fracture of the base of the skull and with signs of high intracranial pressure. Right subtemporal decompression and drainage. Excellent recovery.

No. 061.—Edwin. Five years. White. U. S.

Admitted June 2, 1913. Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Operation June 2, 1913—6 hours after admission. Right subtemporal decompression and drainage.

Discharged June 16, 1913—14 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While playing in the street, patient was knocked down by an automobile; no known loss of consciousness; cried for a period of 30 minutes and then became drowsy; profuse bleeding from the right ear with a small amount of cerebrospinal fluid mixed in the blood; brought to the hospital in the ambulance.

Examination upon admission (4 hours after injury).—Temperature. 100.2°; pulse, 68; respiration, 18; blood-pressure, 116. Well-nourished white child; semiconscious. No signs of shock. Many contusions over face and head; ecchymosis of right orbit and over the right mastoid area. Clotted blood in the right auditory canal. No paralyses. Pupils of normal size and of normal reaction to light. Reflexes—patellar increased but equal; no ankle clonus but a double Babinski; abdominal reflexes absent. Fundi—retinal veins full; definite haziness and edema over the nasal margins and halves of both optic disks. Lumbar puncture—clear cerebrospinal fluid under high tension (approximately 20 mm.) : only small amount of fluid (3 c.c.) allowed to escape for fear of inducing a direct medullary compression. X-ray (Doctor A. J. Quimby)—"no fracture of the skull visible." (This picture was taken while waiting for the operating-room to be prepared.)

Treatment.—An immediate right subtemporal decompression advised to anticipate a possible medullary edema.

Operation (6 hours after admission).—Right subtemporal decompression: usual vertical incision and removal of bone; no complications. Upon incising the dura, which was under much tension, slightly blood-tinged cerebrospinal fluid spurted to a height of 2 inches, and upon enlarging the dural incision, the underlying arachnoid was punctured, allowing the cerebrospinal fluid to spurt to a height of 3 inches, and it continued to do so for almost 4 minutes; a second opening in the arachnoid spurted similarly. (This was the highest pressure I had ever observed in a child having a brain injury.) The cerebrospinal fluid was only slightly blood-tinged and owing to its rapid escape, the bulging cortex became more relaxed and its pulsation visible. Usual closure with one rubber tissue drain inserted beneath the right temporo-sphenoidal lobe into the middle fossa. Duration, 40 minutes.

Post-operative Notes.—No complications. Within 8 hours, the child became more normal mentally and cried for his mother. Profuse drainage of clear cerebrospinal fluid for 36 hours; the drain was then removed.

Examination at discharge (14 days after injury and operation).—Temperature, 98.8°; pulse, 82; respiration, 26; blood-pressure, 118. No complaints except for soreness at the site of operation. Decompression area bulges slightly beyond the flush of scalp; normal pulsation. Otoscopic examination reveals a torn right tympanic membrane in its lower posterior quadrant, hearing of right ear definitely impaired—bone conduction being greater than air conduction. Pupils equal and of normal reaction. Reflexes: active but otherwise negative; no Babinski. Fundi—retinal veins slightly enlarged but no edematous blurring of the details of either optic disk.

Treatment.—Parents continued to keep the child quiet and in a non-excitable condition until the fall—a period of three months; not to play in the sun; light diet of no meat, meat-soup, tea or coffee—but rather vegetables, bread and milk; daily movement of the bowels.

Examination (June 7, 1914—12 months after injury).—No complaints; has been going to school during past winter and "does well." Operative bony opening is being narrowed and almost covered by new bone formation—slightly convex outward. (This occurred in several of the patients due to the periosteum of the bone not being entirely removed but merely scraped back; during the past 3 years, the periosteum is always removed separately and this formation of new bone no longer occurs to any extent.) Hearing of right ear less acute than that of left; bone conduction equals air conduction in right ear. Reflexes negative. Fundi negative.

Examination (September 12, 1916—39 months after injury).—No complaints; "the same as any other child," mother states. Site of operation entirely covered by new bone formation, slightly convex outward. Hearing of right ear equals that of left; air conduction greater than bone conduction in both ears; otoscopic examination negative. Reflexes negative. Fundi negative.

Last Examination (April 20, 1919—70 months after injury).—No complaints; "a strong, well boy in every way." Site of operation entirely covered by new bone formation. Hearing negative. Reflexes negative. Fundi negative.

Remarks.—Several points are of interest in this case-history:

The absence of any blood in the cerebrospinal fluid at lumbar puncture and yet its presence—though of mild degree—in the intracranial cerebrospinal fluid is an observation of rather more frequency than is usually believed; merely because the lumbar puncture obtains clear cerebrospinal fluid is by no means conclusive that an intracranial hemorrhage of even the subdural and subarachnoid type is not present and, at times, in large amount.

The high intracranial pressure in this child is the exception rather than the rule, but its cause being an excess of cerebrospinal fluid producing a "wet," edematous brain rather than a result of profuse hemorrhage, is the usual one—the so-called acute traumatic cerebral edema. It is very doubtful if this degree of high intracranial pressure could have been drained entirely by the natural means of absorption—possibly for an immediate recovery of life but not for an excellent ultimate recovery of normality, which was obtained following the operation. The risk of the operation was slight compared to the great danger of future impairment—emotionally, mentally and physically.

The early recovery of hearing is also the usual result of these injuries in children, in whom the fracture of the petrous bone does not damage the ossicles of the middle ear or the internal ear itself; the tympanic membrane regains its normal appearance very rapidly.

Case 154.—Acute severe brain injury associated with a linear and also a depressed fracture of the left temporo-parietal area and with signs of high intracranial pressure; motor aphasia and right facial weakness. Left subtemporal decompression and drainage. Excellent recovery.

No. 361.—Charles. Ten years. White. School. U. S.

Admitted March 26, 1915. Polyclinic Hospital.

Operation March 27, 1915—14 hours after admission. Left subtemporal decompression and drainage.

Discharged April 12, 1915—14 days after operation.

Family History.—Parents, brothers and sisters are all right-handed, as is the patient.

Personal history negative.

Present Illness.—While playing in street, the boy was struck and knocked down by a taxicab; no loss of consciousness, or if so, only momentarily, but from the time of accident the patient was unable to speak; brought to the hospital in the ambulance.

Examination upon admission (40 minutes after injury).—Temperature, 98.4° ; pulse, 118; respiration, 28; blood-pressure, 110. Conscious, and very restless; understands what is said to him, but he is unable to speak, although he attempts to do so—making inarticulate sounds; he is able to write "you," "yes" or "no," but cannot speak the words; no bleeding from nose, mouth or ears; right and left orbital but no mastoid ecchymoses. Definite tenderness over the left temporo-parietal area where there is a slight depression of the underlying bone of about 2 inches in diameter. Cortical paralysis of right side of face is well marked—with drooping of the right side of the mouth and the disappearance of the right naso-labial fold; right forehead muscles not involved. No paralysis of the arms or legs. Pupils —equal but moderately enlarged and react to light sluggishly. Reflexes: all depressed; no Babinski, Oppenheim or Gordon reflexes; abdominal reflexes absent. Fundi negative.

Treatment.—Vigorous shock measures instituted. Within 10 hours (during the night), the condition of the patient improved but the signs of an increased intracranial pressure became more and more marked.

Examination (12 hours after admission).—Temperature, 99.2°; pulse, 80; respiration, 20; blood-pressure, 118. Conscious but cannot talk; points to his head and holds it as though having a severe headache. No signs of shock. Small hematoma over site of depressed area of bone in the left temporo-parietal area. Otoscopic examination negative. Right facial paralysis of the cortical type persists. No weakness of either arm elicited. Pupils—left slightly larger than right; reaction to light normal. Reflexes: patellar—both exaggerated, right possibly more than left; double exhaustible left ankle clonus but no typical Babinski; abdominal reflexes depressed, right possibly more than left. Fundi—retinal veins dilated; nasal halves of both optic disks obscured by edema. Lumbar puncture — slightly blood-tinged cerebrospinal fluid under high pressure (21 mm.). X-ray (Doctor A. J. Quimby)—"depressed area of bone, 4 cm. in diameter, in left temporal area; an extensive linear fracture of left frontal bone extending backward into left parietal bone and to the upper portion of the depressed area" (Fig. 172).

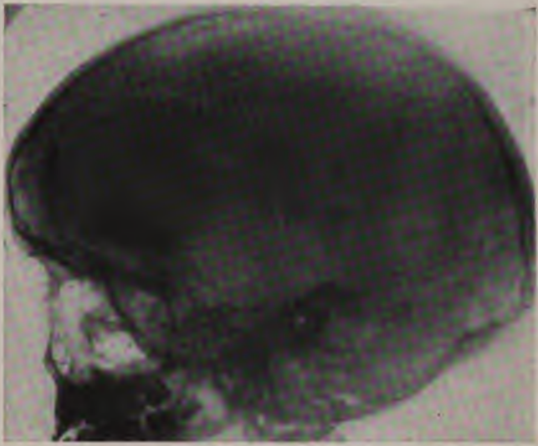


FIG. 172.—Extensive horizontal linear fracture of left vault associated with a depressed fracture, in a patient having a high intracranial pressure associated with a motor aphasia and right facial weakness. Complete recovery following a left subtemporal decompression and removal of bony depression.

Treatment.—An immediate subtemporal decompression advised—both to lower the general increase of the intracranial pressure and also to remove the depressed area of the overlying vault.

Operation (14 hours after admission).—Left subtemporal decompression: usual vertical incision, bone removed, and no complications; bone rather thin and extending to the upper part of the squamous bone was a linear fracture, below which was a depressed area of bone—about 1½ inches in diameter and depressed to a depth of 1 cm. Dura tense and bluish, and upon incising it, bloody cerebrospinal fluid spurted to a height of 8 inches; the underlying cortex tended to protrude but did not rupture, as the lateral ventricle was now punctured successfully and almost 10 c.c. of clear cerebrospinal fluid was removed, permitting the brain to relax and even to pulsate at the end of the operation. No hemorrhage in the cortex itself nor were cortical lacerations visible. Usual closure with 2 drains of rubber tissue inserted. Duration, 42 minutes.

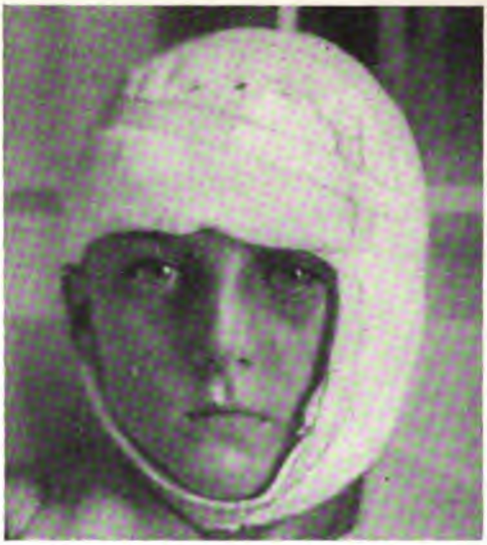


FIG. 173.—The seventh day post-operative, showing the bilateral orbital ecchymoses and the left subconjunctival hemorrhage. The usual dressing and head bandage securely fastened and "anchored" by several small strips of adhesive plaster.

Post-operative Notes.—Uneventful operative recovery; 2 hours after operation, this boy spoke a few words with difficulty and in monosyllables. Within 36 hours after operation, the weakness of the right side of the face lessened and the speech was improved so that sentences could be formed and enunciated clearly. On the seventh day post-operative, a photograph was taken, showing the bandage *in situ* and the orbital ecchymoses (Fig. 173).

Examination at discharge (14 days after operation).—Temperature, 98.6°; pulse, 82; respiration, 22; blood-pressure, 114. No complaints except "I feel a little weak." No facial weakness can be demonstrated by special tests. Speech is negative; patient can repeat: "Around the rugged rock the ragged rascal ran." "The third red riding artillery brigade," and other test phrases, easily and perfectly; spontaneous sentences also enunciated well. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—retinal veins slightly enlarged, but no obscuration of the details of either optic disk. Photograph taken at discharge, showing the area of decompression flush with the scalp (Fig. 174).

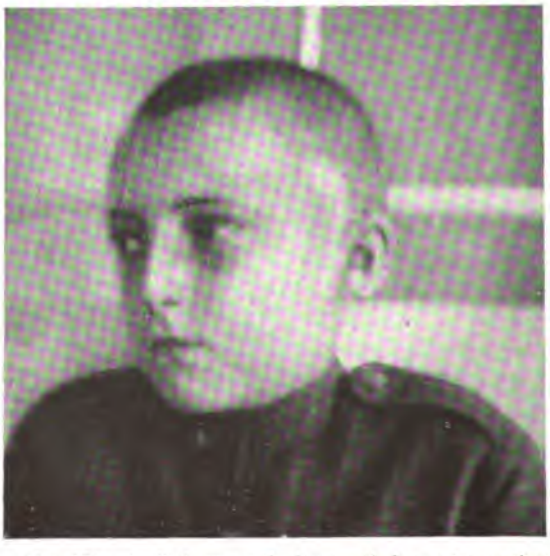


FIG. 174.—At discharge on the fourteenth day post-operative; the left decompression area flush with the surrounding scalp; the orbital ecchymoses persisting.

Examination (September 8, 1917—30 months after injury).—No complaints; "stands near the head of the class." Reflexes negative. Fundi negative. No signs of fa
paralysis nor sp
impairment.

Last Examinati
(February 8, 1919–
months after injury
No complaints. Refl

negative. Fundi negative. Decompression opening has become small from new-bone formation; pulsation hardly palpable.

***Remarks.*—It is doubtful if the depression of the left vault in this patient was sufficient to produce the right facial paralysis and the motor aphasia—and yet it was situated in the position possible to cause these localizing signs of cortical compression. The homolateral pupil was enlarged, however, and thus indicating the paralytic effect of the left supracortical lesion—most probably the supracortical hemorrhage and more or less localized cortical edema—a very common cause for signs of this character.**

It was very interesting to note the signs of an increasing intracranial pressure develop as the symptoms and signs of shock subsided. The fact that the right facial paralysis and aphasia were observed, even when the patient was in shock, would make it appear that the depressed area of the left vault was the cause of these signs, since the supracortical hemorrhage and edema were later developments as exhibited by the signs of an increasing intracranial hemorrhage.

The excellent immediate and ultimate recoveries to be obtained in these children having even severe brain injuries is very gratifying. A longer period of time, however, will be necessary before the end-results are obtainable.

CASE 155.—Acute severe brain injury associated with an increasing intracranial pressure due to a supracortical hemorrhage; Jacksonian convulsive seizures. Left subtemporal decompression and drainage. Excellent recovery.

No. 372.—Robert. Nine years. White. School. U. S.

Admitted April 5, 1915—35 minutes after injury. Polyclinic Hospital.

Operation April 5, 1915—3 hours after admission. Left subtemporal decompression and drainage.

Discharged April 25, 1915—20 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While playing in the street, patient was knocked down by an automobile, striking the left side of his head; no loss of consciousness; brought to the hospital in a semiconscious, drowsy condition.

Examination upon admission (50 minutes after injury).—Temperature, 99.4°; pulse, 78; respiration, 22; blood-pressure, 124. Rather stuporous, but answers questions intelligently; complains of throbbing headache. Left side of face and head, especially the left temporo-frontal area, ecchymosed and boggy from the hematomatous infiltration. No bleeding from nose, mouth or ears; ecchymosis of left orbit, but both mastoid areas of normal appearance. Pupils—left possibly larger than right and of normal reaction to light. Reflexes active, but otherwise negative. Fundi negative.

Treatment.—Expectant palliative treatment; frequent examinations. Within 2 hours, the pulse gradually descended to 60, child became more and more stuporous, until there occurred slight convulsive twitchings of the right side of the face; then a few moments later, a convulsion beginning in the right arm, then the right leg and involving the entire right side of the body, when the convulsion became a typical general epileptiform seizure.

Examination (2 hours after admission).—Temperature, 99.8°; pulse, 58; respiration, 16; blood-pressure, 128. Semiconscious—being aroused with difficulty. Pupils—left pupil slightly dilated, right moderately contracted; sluggish reaction to light. Both eyes turned to the right with nystagmoid twitchings. No weakness of the face elicited nor of the right arm or leg. Reflexes—patellar active, more marked on right; slight ankle clonus on right foot with positive Babinski, Oppenheim and Gordon reflexes; abdominal reflexes depressed but equal. Fundi—dilated retinal veins with an edematous blurring of the nasal halves of both optic disks. Lumbar puncture—bloody cerebrospinal fluid under high intracranial pressure (approximately 20 mm.).

Treatment.—An immediate left subtemporal decompression and drainage advised.

Operation (3 hours after admission).—Left subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications. No line of fracture ascertained. Dura bluish and under high tension; upon incising it, dark blood spurted to a height of 4–5 inches and continued to spurt for several seconds. Upon enlarging dural opening, the suffused and congested cortex tended to protrude, but it did not rupture owing to the rapid escape of much supracortical blood and cerebrospinal fluid; much subarachnoid blood in the sulci. An attempt to puncture the lateral ventricle was not successful in an effort to lower the high cortical tension, which was eventually lessened by the drainage of much free blood and excess cerebrospinal fluid, so that normal cortical pulsation now became visible. No cortical hemorrhage or lacerations ascertained. Usual closure with two drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Uneventful operative recovery; much drainage of blood continued for 2 days and then the drains were removed. Child became conscious within 5 hours after the operation and his general condition was markedly improved in that the pulse ascended to 74 and no convulsions occurred after the operation; during the operation, the child had several convulsions similar to the one before operation and always beginning in the right side of the face, but none was severe. At the close of the operation, extreme restlessness with frequent twitchings of the facial muscles appeared; a restraining sheet was required and also sedatives; he became quiet after 1½ hours and consciousness returned after 5 hours, when he was able to answer questions; he remembered the circumstances of the accident very well.

Examination at discharge (20 days after operation).—Temperature, 98.8°; pulse, 80; respiration, 22; blood-pressure, 118. No complaints other than a soreness of the left side of head; no headache. Perfectly normal mentally. No convulsions since the operation. Pupils equal and react normally. Reflexes: active but otherwise negative; no Babinski, although a possible tendency to a right Babinski. Fundi—retinal veins slightly enlarged; indistinct blurring of the lower nasal quadrants of both optic disks. X-ray report (Doctor A. J. Quimby)—"no line of fracture visible."

Treatment.—Parents cautioned regarding a non-proteid diet for the patient and general hygienic rules.

Examination (September 4, 1917—29 months after injury).—No complaints; "possibly a little more cranky than the other children." Pupils negative. Reflexes active but otherwise negative. Fundi negative.

Last Examination (April 20, 1919—48 months after injury).—No complaints; goes to school daily. Decompression area depressed; normal pulsation. Reflexes negative. Fundi negative.

Remarks.—This case-history is instructive in that the patient was perfectly conscious upon admission to the hospital—in fact, there had been no loss of consciousness following the cranial injury—and then he gradually exhibited the signs of an increasing intracranial pressure. It is fortunate he was not discharged from the hospital at the time of admission, as being merely a trivial "bump" on the head—a not infrequent catastrophe.

The absence of a fracture of the skull to be disclosed, either by the usual signs or the röntgenograms, is of no significance other than to emphasize the comparative unimportance of the presence or not of a fracture of the skull in these patients having brain injuries.

A lumbar puncture should have been performed upon the patient at the time of the first examination, and the presence of blood in the cerebrospinal fluid would have impressed us more with the seriousness of the patient's condition—from the standpoint that a definite intracranial injury had occurred.

The localized convulsive twitchings and eventually epileptiform seizures indicating a left cortical lesion, together with the homolateral dilatation of the left pupil indicating the paralytic effect of the left supracortical hemorrhage, whereas the right pupil became constricted as the result of the irritative lesion over the right cerebral cortex—these signs are both characteristic and instructive.

Case 156.—Acute severe brain injury associated with an increasing intracranial pressure due to subdural hemorrhage and cerebral edema; aphasia and left facial paralysis. Right subtemporal decompression and drainage. Excellent recovery.

No. 887.—Gustave. Six years. White. School. U. S.

Admitted Sept. 27, 1917—40 minutes after injury. Polyclinic Hospital.

Operation September 29, 1917—40 hours after admission. Right subtemporal decompression and drainage.

Discharged October 17, 1917—18 days after operation.

Family history negative. Grandfather, mother and one brother are left-handed.

Personal history negative. Patient is right-handed.

Present Illness.—While playing upon a fire-escape, child fell to the ground below—a distance of 45 feet; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (50 minutes after injury).—Temperature, 97.8°; pulse, 138; respiration, 30; blood-pressure, 98. Unconscious and in severe shock. Ecchymosis of right orbit. No bleeding from nose, mouth or ears; no mastoid ecchymosis. No paralyses ascertained. Pupils widely dilated. Reflexes abolished. Fundi negative.

Treatment.—Vigorous shock measures instituted—especially heated blankets, hot water bottles, and rectal enemata of hot black coffee. After 6 hours, the general condition gradually improved, but the signs of moderate shock persisted for over 28 hours.

Examination (30 hours after admission).—Temperature, 99° ; pulse, 78; respiration, 78; blood-pressure, 116. Semiconscious, although at times patient can be aroused sufficiently to turn over, look around, but not to answer questions; he attempts to speak but cannot; he will, however, make signs with his right hand and with his head; apparently, a definite motor aphasia but not a sensory one since he can read questions and also hear them; no astereognosis nor apraxia. Definite left facial paralysis of the cortical type (left forehead and upper third of left side of face not involved). Left arm and left leg not appreciably weaker than right arm or right leg. Pupils of equal size (right possibly smaller than left) and of normal reaction to light. Reflexes—very active but otherwise negative; no Babinski. Fundi —retinal veins full; nasal halves and temporal margins of both optic disks blurred by edema. Lumbar puncture—bloody cerebrospinal fluid under high pressure (20 mm.).

Treatment.—The expectant palliative treatment was continued during the night for 8 hours, when an immediate operation was advised as being the safer procedure. No röntgenogram was taken before the operation.

Operation (40 hours after admission).—Right subtemporal decompression: usual vertical incision, bone removed, and no complications; in the upper posterior portion of the squamous bone was a linear fracture extending into it. Dura was very tense and bluish, and upon incising it bloody cerebrospinal fluid spurted a distance of 2 cm. The underlying cortex was swollen and edematous with multiple punctate hemorrhages in it: much bloody cerebrospinal fluid escaped, allowing the cortex to recede and to pulsate. No large cortical hemorrhage or laceration observed. Usual closure with two drains of rubber tissue inserted. Duration, 35 minutes.

Post-operative Notes.—Uneventful operative recovery; child became perfectly conscious within 36 hours after operation, the weakness of the left side of face lessened and he was able to talk in monosyllables on the fourth day: sitting up in bed on the sixth day. A röntgenogram taken on the tenth day post-operative disclosed a vertical linear fracture of the left parietal bone descending into the left squamous bone; the right decompression opening with four silver clips *in situ* is clearly pictured (Fig. 175).

Examination at discharge (18 days after operation).—No complaints other than an occasional headache. Decompression area flush with the scalp; normal pulsation. No facial weakness elicited. No impairment of speech but an apparent retardation of thought (possibly natural for the child since the parents do not notice it). Hearing negative; otoscopic examination negative. Pupils equal and react normally. Fundi—retinal veins slightly enlarged; indistinct edematous blurring of lower section of nasal margins of both optic disks.

Treatment.—Parents cautioned regarding general hygienic rules and a non-proteid diet; not to return to school until the spring.

Examination (May 16, 1918—8 months after injury).—No complaints: has been attending school since February; does well in school. Decompression area slightly depressed and pulsates normally. Reflexes active but otherwise negative. Fundi negative.

Last Examination (April 10, 1919—19 months after injury).—No complaints referable to the head. Operative area depressed and only slight pulsation observed due to narrowing of bony opening from new-bone formation about the periphery. Reflexes negative. Fundi negative.

Remarks.—As the severity of the shock subsided, the gradual onset of the left facial paralysis of the cortical type, with the motor aphasia occurring in a right-handed child but whose brother, mother, and grandfather were left-handed, indicated a greater lesion of the right cerebral cortex, especially the lower motor area; the ipsolateral constriction of the right pupil tended to confirm this, but there were no marked changes in the reflexes to strengthen this belief. The signs of the high intracranial pressure made necessary the operation of decompression and drainage and naturally on the right side, although it would have been better judgment to have operated 8 hours earlier instead of permitting the patient to run the risk of extreme medullary compression during the night.

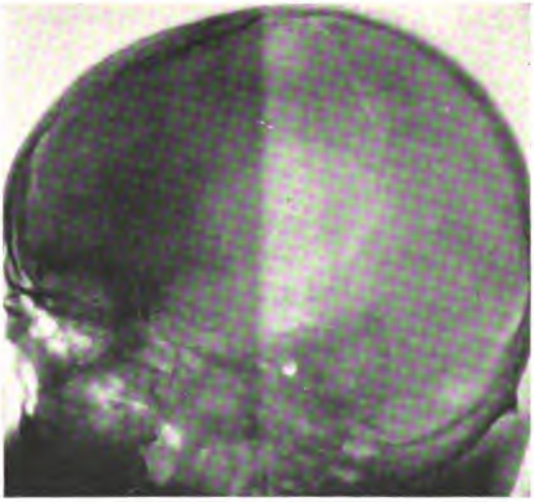


FIG. 175.—Extensive linear fracture of left vault in a patient having a motor aphasia and left facial paralysis of the cortical central type. Complete recovery following a right decompression and drainage of the subdural hemorrhage and cerebral edema.

The motor speech centre being on the right side in this patient would tend to confirm the opinion that at least in early life the situation of the motor speech centre in either cerebral cortex is one of heredity rather than whether the patient himself is right- or left-handed; possibly later in this patient, who was considered to be right-handed, the speech centre may have developed in the left cerebral cortex, but I doubt it; possibly both cerebral hemispheres were equally well developed for the function of speech—one being latent.

The absence of convulsive twitchings or seizures in this patient cannot be explained, except that patients having similar irritative supracortical lesions, vary in their resistance to convulsions—whether a greater nerve-cell stability or not, is not known; patients having the same irritative cortical lesion (as well as can be ascertained at operation or at autopsy) may or may not have convulsions and epileptiform seizures.

After the shock had subsided, the increasing intracranial pressure made the operation of decompression and drainage the safer procedure, and it was of comparatively no importance whether a fracture of the skull was present or not—the operative indications were the same.

Case 157.—Acute severe brain injury associated with a subdural hemorrhage, cerebral edema and with a definite paraplegia; a marked increase of the intracranial pressure. Right subtemporal decompression and drainage. Excellent recovery.

No. 899.—Katherine. Three years. White. U. S.

Admitted October 7, 1917—4 weeks after injury. Polyclinic Hospital.

Operation October 7, 1917—10 hours after admission. Right subtemporal decompression and drainage.

Discharged October 22, 1917—15 days after operation.

Family history negative.

Personal history negative.

Present Illness.—Four weeks ago, while in the best of health, child fell from a baby carriage down upon the brick pavement four steps below—a distance of 5 feet, and striking upon the back of the head; no loss of consciousness; child cried, was able to get up and walk away; she was put to bed, however, within one-half hour, and upon the following morning (14 hours later), it was found that the child could not move the lower legs below the knees; 24 hours later, both legs were entirely paralyzed. Child remained in a semiconscious condition with no marked increase of temperature beyond 99.8°; child complained, however, of headaches and was at times very restless. Patient has been treated by the expectant palliative method by the family doctor during the past 4 weeks since the injury, but the condition of the child has remained practically the same; no convulsions have occurred.

Examination upon admission (4 weeks after injury).—Temperature, 99°; pulse, 80; respiration, 24; blood-pressure, 106. Rather stuporous and drowsy; upon being aroused, however, child will answer "yes" and "no" and complains of headache. No external evidence of cranial injury except a slight tenderness over the occipital prominence. No stiffness nor rigidity of the neck and no positive Kernig elicited nor ocular paralyses; no strabismus nor nystagmoid twitches ascertained. No orbital nor mastoid ecchymoses. Hearing negative; otoscopic examination negative. The legs could not be moved below the knees and they were definitely weak upon flexing and extending the thighs upon the pelvis; an indefinite hypesthesia over both legs with no marked signs of demarcation upon approaching the pelvis; this sensory impairment more marked below the knees. (Owing to the drowsy and stuporous condition of the child, a careful sensory examination could not be made and the accuracy of our findings is doubtful.) The sense of position of the toes apparently impaired. Child would cry upon being disturbed and then again fall asleep during these examinations. Pupils equal and react to light normally. Reflexes—patellar very much exaggerated but equal; inexhaustible ankle clonus and a typical bilateral Babin abdominal reflexes both absent. Fundi: retinal veins full, tortuous an places buried in edematous retinæ; the details of both optic disks obse by edema—there being present a double papilledema of one diopter swel Lumbar puncture—straw-colored cerebrospinal fluid under an incre pressure (16 mm.); 6 c.c. removed for examination; (bacteriological r by Doctor Jeffries)—"numerous degenerated red blood-cells; no ba found"; later report of culture—"negative for bacteria." X-ray report (Doctor G. W. Welton)—"no fracture of the skull observed." Urine examination negative.

Treatment.—The presence of the high intracranial pressure with a definite weakness of both legs persisting for a period of 4 weeks following a cranial injury in spite of the expectant palliative method of treatment, indicated the necessity of a mechanical relief of the intracranial condition in order to avoid further complications and to give the child her best chance of recovery. Considering the injury merely as an incident and not the cause of the condition, the case-history is very similar to that of a mild meningitis and usually of the tuberculosis type occurring in children, and yet the absence of the signs of meningeal irritation and the negative cerebrospinal fluid tended to exclude this possibility. An immediate right subtemporal decompression and drainage was advised, but it was not performed for several hours in order to obtain the written consent of the parents; the condition of the patient, however, remained practically the same.

Operation (4 weeks after injury).—Right subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications. Dura very tense, and upon incising it straw-colored cerebrospinal fluid spurted to a height of 8 inches and continued to do so for almost 1 minute; upon enlarging dural opening, the underlying "wet," edematous and congested cortex tended to protrude, and if a large quantity of cerebrospinal fluid had not escaped, it is feared that the cerebral cortex would have ruptured on account of the high intradural pressure; fortunately, the cortex became less tense and finally pulsated feebly at the end of the operation. No gross supracortical or cortical hemorrhage observed and no cortical lacerations—merely a very "wet," edematous cortex with a reddish blue collection of old free blood in the sulci about the cortical veins, where it was becoming organized and would later undoubtedly present the picture of a whitish induration about the cortical veins, as is observed in the chronic cases of supracortical hemorrhage. Usual closure with 2 drains of rubber tissue inserted. Duration, 35 minutes.

Post-operative Notes.—The excellent and almost immediate improvement observed in this patient is remarkable; the child appeared much brighter upon the day following the operation, became more interested in things and no longer complained of headache. She was able to move the toes upon the third day after operation and within a week she could move both legs awkwardly. All drainage of straw-colored cerebrospinal fluid ceased 30 hours after operation and the drains were then removed. The operative incision healed *per primam*. Twelve days after operation, the child was able to stand alone and to walk several steps when supported. The parents now insisted upon taking the child home.

Examination at discharge (15 days after operation).—Temperature, 98.8°; pulse, 84; respiration, 26; blood-pressure, 110. Patient no longer complains of headache. Operative area bulges but pulsates normally. The child can now walk several steps without being supported and shows a daily improvement; no sensory impairment can be elicited. Hearing negative. No ocular paralyses. Pupils equal and react normally. Reflexes—patellar very active but equal; right exhaustible ankle clonus but no Babinski can be elicited, though no plantar flexion can be obtained upon the right foot; abdominal reflexes depressed but equal. Fundi—retinal veins enlarged but not buried in edema; nasal margins of both optic disks obscured but the other details are distinct.

Treatment.—Parents cautioned to be very careful regarding the child's diet and her general hygienic life; daily massage for the legs advised.

Examination (May 18, 1918—7 months after operation).—No complaints except that the child becomes more easily tired than other children; no headaches, however, and is apparently well. Decompression area almost flush with the surrounding scalp; normal pulsation. No weakness of either leg can be elicited; no sensory impairment. Reflexes active but otherwise negative. Fundi—retinal veins possibly still larger than normal; all details of both optic disks clear and distinct.

Last Examination (February 6, 1919—16 months after operation).—No complaints. "A well girl." Operative area slightly depressed below flush of scalp; normal pulsation. Reflexes active but otherwise negative. Fundi negative.

Remarks.—In the absence of a straw-colored cerebrospinal fluid with numerous red blood-cells in it, the diagnosis could very easily have been a mild meningitis and most probably of tuberculous character; so frequently, the history of a preceding cranial injury is obtained from the parents of these children, and yet this history of a cranial injury must not be carelessly excluded unless careful examinations do not show, and they did in this child, that the cranial injury was not merely an incident but rather the cause of the intracranial lesion. A longer period of time, however, must elapse before it can be definitely stated that the recovery of normality is a permanent one, and that there will be no later signs of the intracranial condition—especially epilepsy in its various forms.

It was rather interesting to observe the persistency of the right ankle clonus in its exhaustible form and also the right Babinski, in that no plantar flexion could be obtained and these signs upon the right foot controlled by the left cerebral hemisphere, which was not decompressed as much as the right cerebral hemisphere—the operation being a right subtemporal decompression. It would appear that the acute cerebral edema was drained more from the right cerebral cortex than from the left cerebral cortex, and this would naturally be expected—the operation being over the right cerebral hemisphere. If the decompression area had remained tense and bulging for a period longer than one week, then it probably would have been advisable to have performed a left subtemporal decompression and drainage in order that the greatest ultimate recovery could be obtained.

It is surprising that no convulsive seizures occurred as the result of cortical irritation of the supracortical free blood and the presence of increased intracranial pressure; it is possible that this complication have occurred later unless relieved as in this patient.

Case 158.—Acute severe brain injury associated with a fracture o vault and of the base of the skull and with a supracortical hemorrhage an increased intracranial pressure; left hemiplegia and Jacksonian c sive seizures. Right subtemporal decompression and drainage. Excellent recovery.

No. 933.—James. Seven years. White. School. U. S.

Admitted December 23, 1917. Polyclinic Hospital.

Operation December 29, 1917—6 days after admission. Right subtemporal decompression and drainage.

Discharged January 12, 1918—13 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While playing in a snow-pile in the street, child was knocked down by an automobile; loss of consciousness for several minutes; brought to the hospital in the ambulance.

Examination upon admission (10 minutes after injury).—Temperature, 97.2°; pulse, 124; respiration, 32; blood-pressure, 98. Semiconscious and in severe shock. Slight contusion of the scalp and a diffuse hematoma over the right parietal eminence. Profuse bloody discharge mixed with cerebrospinal fluid from the right auditory canal; clotted blood observed in both external nares. Right orbital and right mastoid ecchymoses. No paralyses nor sensory impairments ascertained. Pupils dilated and react to light sluggishly. Reflexes all depressed and no Babinski obtained. Fundi negative. No further neurological examination was made at this time owing to the severe degree of shock.

Treatment.—Vigorous shock measures instituted—particularly external heat by warm blankets and hot water bags, repeated rectal enemata of hot black coffee—3 ounces every 2 hours—and absolute rest and quiet. Within 8 hours, the child rapidly recovered from the severe condition of shock and as the general condition was good with no marked signs of a severe intracranial lesion, it was believed that the child would make an excellent recovery under the expectant palliative treatment alone. No signs of a high intracranial pressure appeared, although at a lumbar puncture 40 hours after admission, bloody cerebrospinal fluid was obtained under an increased pressure of 11 mm.; 12 c.c. were slowly removed as a means of drainage therapeutically, and this was again repeated upon the fourth day after admission, when the pressure was found to be only 10 mm. and this time only 10 c.c. of blood and cerebrospinal fluid were removed. The discharge of blood and cerebrospinal fluid from the right ear ceased within 12 hours after admission; an otoscopic examination at this time disclosed a rupture of the lower posterior portion of the right tympanic membrane.

The child remained in a drowsy stuporous condition—although at times he was very restless—for a period of 5 days, and as the general condition remained good and there developed no definite signs of an increasing intracranial pressure or other local signs of cerebral impairment, the expectant palliative treatment was continued. On the morning of the sixth day after admission, the child had a convulsive seizure beginning in the left side of the face, then the left arm, and finally the left leg—continuing for almost one minute, when a general convulsive seizure occurred and lasted 3 minutes; there was incontinence of both urine and feces and a left hemiparesis was observed following the convulsion. The following examination was now made:

Examination (6 days after admission).—Temperature, 99.4°; pulse, 80; respiration, 22; blood-pressure, 112. Very drowsy and stuporous; upon being aroused, however, patient can reply to questions in a confused manner. Definite weakness of the entire left side of body—including left side of face and of the cortical type of paralysis in that the muscles of the left forehead were not involved; no definite sensory impairment elicited (patient was so drowsy that the tests of astereognosis and apraxia could not be utilized). Pupils—right larger than left and reacts to light possibly more sluggishly than left. (At this point of the examination, spasmodic twitchings of the left side of the face occurred, especially about the left side of the mouth and left eye; no loss of consciousness was apparent.) Reflexes—patellar very active, left more than right; exhaustible ankle clonus and a characteristic left Babinski, while there was a tendency to a right Babinski; abdominal reflexes could not be elicited. Fundi—retinal veins full; nasal half of right optic disk obscured by edema to a greater degree than in the left optic disk. Lumbar puncture—straw-colored cerebrospinal fluid under increased pressure (14 mm.). X-ray report (Doctor G. W. Welton)—"a vertical line of fracture extended downward toward the right mastoid area from the right parietal bone in its posterior portion" (Fig. 176).

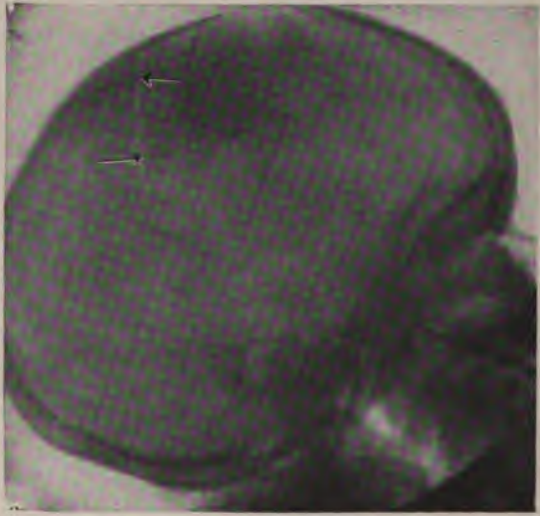


FIG. 176.—Vertical linear fracture of right vault in a patient having a high intracranial pressure with resulting left hemiparesis and Jacksonian convulsive seizures. Excellent recovery following a right subtemporal decompression and drainage of the supracortical hemorrhage.

Treatment.—In the presence of the Jacksonian convulsive seizure with repeated left facial twitchings and an increasing intracranial pressure associated with a left hemiparesis, it was considered advisable to perform a right subtemporal decompression and drainage in the hope that the intracranial condition could be relieved.

Operation (6 days after admission).—Right subtemporal decompression and drainage (primary anesthesia and at intervals): usual vertical incision, bone removed, and no complications. Dura bluish and under moderate tension; upon incising it much straw-colored cerebrospinal fluid escaped, revealing a very "wet," swollen, congested cortex; at the upper portion of the dural opening, a small layer of supracortical hemorrhage was observed, and this currant-jelly clot was removed carefully by means of the spoon-spatula.

The bulging cerebral cortex now receded and pulsated almost normally. No cortical laceration or cortical hemorrhages observed. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Four hours after the operation, a slight convulsive twitching of the left side of the face occurred and this was the last spasmodic contraction observed; the child made an excellent recovery in that the weakness of the left side of the body had entirely disappeared within 50 hours after operation, the incision healed *per primam,* and no further convulsive seizures occurred.

Examination at discharge (13 days after operation).—Temperature, 98.8°; pulse, 82; respiration, 24; blood-pressure, 112. No complaints. "I am all right." Operative area flush with the surrounding scalp and pulsates normally. No weakness of the left side of body can be elicited by special tests; no sensory impairment; no astereognosis nor apraxia revealed. Hearing of right ear impaired; bone conduction greater than air conduction. Pupils of equal size and of normal reaction. Reflexes: patellar active but apparently equal; no ankle clonus nor Babinski; abdominal reflexes—left possibly less active than right. Fundi—retinal veins slightly enlarged; an indistinct blurring of the lower portions of the nasal margins of both optic disks.

Examination (September 20, 1918—9 months after injury).—No complaints; no convulsive seizures since leaving the hospital and the child attended school during the spring. No weakness of the left side of the body can be ascertained by special tests. Reflexes active but otherwise negative. Fundi negative. Operative area is depressed and slight pulsation palpable. Hearing of the right ear less acute than that of left—bone conduction still being greater than air conduction.

Last Examination (May 18, 1919—19 months after the injury).—No complaints referable to the former head injury; child is doing well in school and no convulsion has occurred; patient is no more irritable or restless than the other children. Hearing of right ear possibly less acute than that of left; bone conduction, however, is not greater than air conduction; otoscopic examination of right tympanic membrane is negative, except for a slight thickening of its lower posterior portion—possibly a little scar tissue; no retraction of the drum observed. Reflexes active but otherwise negative. Fundi negative.

Remarks.—It is possible if the repeated lumbar punctures and drainage had been continued daily, that the complication of convulsive seizures with the left hemiparesis could have been prevented and the operation thus avoided; as the intracranial pressure, however, did not increase, nor were there any signs of it being increased, naturally the lumbar punctures were not continued, and it was only when the twitchings of the left side of the body occurred and followed by the left hemiplegia, that the severity of the condition was recognized and an immediate right subtemporal decompression and drainage advised. Although the intracranial pressure was not high—it being only 14 mm. at operation—yet the irritative presence of the supracortical hemorrhagic clot was sufficient to cause the left Jacksonian convulsions and the subsequent left hemiparesis, and it was this complication that necessitated an early relief of the intracranial condition—and best accomplished by a right subtemporal decompression and drainage—the operation of choice.

The excellent recovery of normality with no recurrence of the convulsive seizures is very favorable and gratifying, and yet a longer post-operative period must elapse before this patient can be considered as beyond all danger; his life should be a well-regulated hygienic one with the avoidance of all mental and emotional strain; naturally, the diet and the avoidance of alcohol are important. The rapid improvement of the hearing of the right ear is characteristic of these cases in children.

CASE 159.—Acute severe brain injury associated with a fracture of the vault and with a subdural hemorrhage; an increasing intracranial pressure. Left subtemporal decompression and drainage. Excellent recovery.

No. 983.—William. Ten years. White. School. U. S.

Admitted May 20, 1918. Polyclinic Hospital.

Operation May 21, 1918—20 hours after injury. Left subtemporal decompression and drainage.

Discharged June 5, 1918—15 days after admission.

Family history negative.

Personal history negative.

Present Illness.—While crossing the street, patient was struck by a boy riding a bicycle; knocked down, striking the cement curbing with the left side of his head; no loss of consciousness and was able to walk to the accident room of the hospital with the aid of several boy friends.

Examination upon admission (30 minutes after injury).—Temperature, 97.2°; pulse, 104; respiration, 20; blood-pressure, 102. Rather stuporous and drowsy and in shock. Small contusion of the scalp overlying the left fronto-parietal area; marked tenderness elicited upon palpating this area. No bleeding from nose, mouth or ears, but during the examination he vomited twice—almost projectile in character. Pupils slightly enlarged and react to light sluggishly. Reflexes—all depressed; no Babinski. No extensive examination made at this time on account of the condition of severe shock—the patient being immediately admitted to a ward bed.

Treatment.—Vigorous shock measures instituted and the usual expectant palliative treatment—especially external warmth and hot water bottles, ice-helmet and hot black coffee per rectum every two hours in amounts of 3 ounces; within 4 hours, the condition of the child improved in that the temperature gradually ascended to normal, the blood-pressure to 110, while the pulse- and respiration-rates descended more to normal and became more regular. After 16 hours, however, the pulse had descended to 72, while the child had become more stuporous and almost semiconscious; when aroused, the patient would complain of severe headache.

Examination (18 hours after admission).—Temperature, 101°; pulse, 70; respiration, 20; blood-pressure, 116. Very stuporous and difficult to arouse. Slight left orbital ecchymosis but no mastoid ecchymoses. No paralysis nor marked impairment of sensation ascertained. Otoscopic examination negative. Pupils—both small, with little or no reaction to light. *Reflexes*—patellar very active, right possibly more than left; no ankle clonus nor Babinski, but no plantar flexion obtained upon the right foot; abdominal reflexes depressed but equal. Fundi—retinal veins full and slightly tortuous; nasal halves of both optic disks obscured by edema and slight blurring of the temporal margins of both optic disks. Lumbar puncture—blood-tinged cerebrospinal fluid under high intracranial pressure (18 mm.); 4 c.c. allowed to escape very slowly and only a very small quantity (for fear of precipitating medullary complications). X-ray (Doctor G. W. Welton)—"an extensive linear fracture of the vault involving the left parietal and the left portion of the frontal bone where it terminates in a fork; also a linear fracture of the right parietal bone" (Fig. 177).

Treatment.—The expectant palliative treatment was continued until permission for the operation could be obtained from the parents one hour later; condition remained practically the same, except that the pulse had descended to 68 and the child had bebecome even more stuporous.

Operation (20 hours after injury).—Left subtemporal decompression and drainage (primary anesthesia and at intervals as upon opening the dura, etc.): usual vertical incision, bone removed, and no complications. Dura tense, slightly bluish and bulging; a small extradural clot was removed from the anterior upper area of the dural exposure. Upon incising the dura, bloody and straw-colored cerebrospinal fluid spurted to a height of 5 inches, and upon enlarging dural opening much free blood and cerebrospinal fluid escaped, permitting the "wet," edematous cortex to recede and to pulsate slightly. No cortical lacerations or hemorrhages observed—merely a very "wet" and congested swollen brain. Usual closure with two drains of rubber tissue inserted. Duration, 40 minutes.

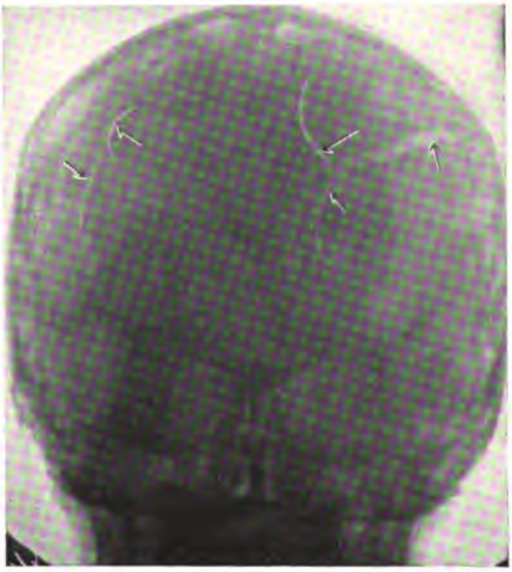


FIG. 177.—Two linear fractures of the parietal areas in a patient having a subdural hemorrhage with an increasing intracranial pressure. Excellent recovery following a left subtemporal decompression and drainage.

Post-operative Notes.—Child became fully conscious within 8 hours after operation, cried for his mother and said, "I feel all right" and "I want to go home"; pulse ascended to 88 and the ophthalmoscope revealed a lessening of the obscuration of both optic disks. Profuse drainage of blood-tinged and straw-colored cerebrospinal fluid continued for 24 hours and then practically ceased, so that both drains were then removed. The operative incision healed *per primam*—all skin sutures being removed upon the sixth day.

Examination at discharge (14 days after operation).—Temperature, 98.8°; pulse, 80; respiration, 24; blood-pressure, 112. No complaints except for a general soreness over left side of head; slight tenderness along the lines of fracture as revealed by the röntgenogram. Decompression area protrudes slightly beyond the flush of scalp; normal pulsation. Hearing negative. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—retinal veins slightly enlarged; lower nasal margins of both optic disks not entirely distinct.

Treatment.—Patients advised to restrict the child's activities during the coming summer and to observe the usual hygienic rules of diet, sleep and daily regulation of the bowels.

Examination (September 4, 1918—4 months after injury).—No complaints and parents wish to know regarding the advisability of the boy going to school. Operative area slightly depressed beneath the flush of scalp and pulsates normally. No retardation mentally nor an emotional instability elicited. Reflexes active but otherwise negative. Fundi negative. Parents advised to permit the boy to attend school, but to caution him and the teacher regarding a too strenuous exertion—no examinations for at least a year.

Last Examination (May 14, 1919—12 months after injury).—No complaints and child has done well in school; no headache and no visual impairment. Reflexes active but otherwise negative.

Remarks.—The presence of an increasing intracranial pressure to the height of 18 mm. as registered by the spinal mercurial manometer, with an increasing stupor and the definite signs of an intracranial pressure as disclosed by the ophthalmoscope—these findings made advisable the early mechanical lowering of this increasing intracranial pressure, whether due to hemorrhage or an excess amount of cerebrospinal fluid. The necessity for the operation is less indicated in children than in adults—all other factors being the same, but it is in this type of patient where the intracranial pressure is rapidly increasing that it is most dangerous to delay the operation of decompression until the extreme signs of medullary compression, such as a very much lowered and irregular pulse- and respiration-rate, profound unconsciousness and a height of intracranial pressure, even to the degree of papilledema or even measurable swelling of the optic disks and an increased pressure of the cerebrospinal fluid to 20 mm. and even higher. It is in these patients having a high intracranial pressure that repeated lumbar punctures and drainage therapeutically would be most dangerous for fear of producing an acute medullary compression and its subsequent medullary edema.

The unimportance of the presence or not of a linear fracture of the vault in this case is well demonstrated; if the underlying dura had been torn, it would have been possible for much of the subdural hemorrhage and excess cerebrospinal fluid to have escaped into the extracranial tissues of the scalp to form a hematoma, and in this manner the increasing intracranial pressure might have been sufficiently lowered to have avoided the necessity operation and therefore the fracture in itself would have been a favo occurrence in this case; as it was, however, the presence of the fra was merely an incident rather than an important factor in the con both from the standpoint of diagnosis and of prognosis, and especi the treatment of the intracranial condition.

Case 160.—Acute severe brain injury associated with a fracture of the vault and with an extradural hemorrhage due to a rupture of the middle meningeal artery; a definite increase of the intracranial pressure. Left subtemporal exploration and ligation of the middle meningeal artery. Excellent recovery.

No. 1013.—Morris. Nine years. White. School. U. S.

Admitted September 28, 1918. Polyclinic Hospital.

Operation September 29, 1918—17 hours after admission. Left subtemporal exploration.

Discharged October 13, 1918—15 days after injury.

Family history negative.

Personal history negative. Patient and relatives are all right-handed.

Present Illness.—While crossing the street, patient was struck by an autotruck and knocked down; carried into a drug-store in a semiconscious condition; brought to the hospital in the ambulance.

Examination upon admission (45 minutes after injury).—Temperature, 98.6°; pulse, 130; respiration, 30; blood-pressure, 104. Rather stuporous and in mild degree of shock. Over the left temporo-parietal area near the anterior portion of the left parietal crest is a tense fluctuating hematoma of the size of a lemon; this entire area very tender upon palpation. Left orbit ecchymosed and multiple contusions over the left side of the scalp and the body. No paralyses or marked sensory impairments ascertained. No bleeding from the nose, mouth or ears; no mastoid ecchymoses. Pupils equal and react normally to light. Reflexes—present and equal; no Babinski. Fundi negative. No extensive examination performed at this time for fear of prolonging and even increasing the general condition of shock.

Treatment.—Expectant palliative. Within two hours, the general condition had so improved in that the pulse- and respiration-rates had descended to 96 and 24, respectively, that a lumbar puncture was performed and clear cerebrospinal fluid was obtained under only a slightly increased pressure (9 mm.) and 10 c.c. were slowly withdrawn. The hematoma over the left parietal crest, having become more tense and even larger than upon admission, it was considered advisable to aspirate it through a clean area of the scalp and with careful asepsis; almost 3 ounces of pure blood were removed and a tight bandage was applied. Within 6 hours, however, the hematoma had refilled and it was again aspirated and almost 4 ounces of pure blood were this time removed and another tight bandage applied. The X-ray report (Doctor G. W. Welton) was "a wide linear fracture extending obliquely from the left parietal crest downward and forward into the left frontal bone just above the left external angular process" (Fig. 178), and it was therefore feared that the left middle meningeal artery had been torn by the line of fracture and that the resulting extradural blood was escaping through the line of fracture into the extracranial tissues of the scalp to form the hematoma; this belief was further strengthened by the fact that the hematoma again became tense and of its former large size for the third time within another period of 4 hours.

Examination (15 hours after admission).—Temperature, 99.6°; pulse, 88; respiration, 24; blood-pressure, 112. Conscious but drowsy; talks rationally and answers questions with no mental or emotional confusion. Hematoma has become tense again for the fourth time and very tender. Left orbit closed by edema. A slight weakness of the right side of the face and of the right arm has appeared within the last four hours; no definite impairment of the right leg; no sensory disturbance. No aphasia elicited by special tests. Hearing negative. Otoscopic examination negative. Pupils—left possibly larger than right; reaction to light normal. No ocular paralyses; no nystagmus. Reflexes—patellar exaggerated, right more than left; no ankle clonus but suggestive right Babinski; abdominal reflexes depressed but no inequality elicited. Fundi: retinal veins enlarged—left possibly more than right; nasal margins of both optic disks and nasal half of left optic disk blurred by edema. Lumbar puncture—clear cerebrospinal fluid under increased pressure (14 mm.).

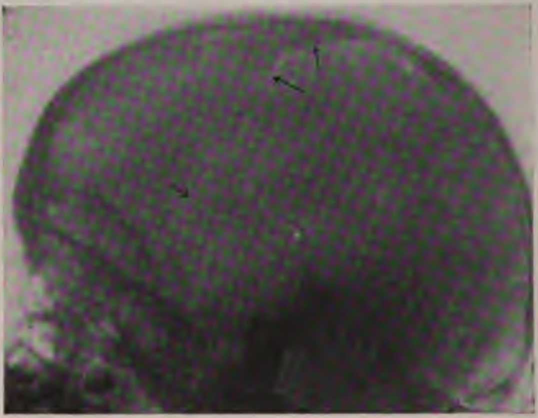


FIG. 178.—A wide extensive linear fracture of the left vault, causing a rupture of the left middle meningeal artery. Excellent recovery following a left subtemporal exploration, removal of extradural hemorrhage and the ligation of the left middle meningeal artery.

Treatment.—For fear that an extradural hemorrhage was compressing the left cerebral cortex, a left subtemporal exploration was advised to be performed immediately.

Operation (17 hours after admission).—Left subtemporal exploration, removal of extradural hemorrhage and ligation of left middle meningeal artery: usual vertical incision, removal of bone, and no complications; upon enlarging the bony opening as made by the Doyen perforator and burr, a dark currant-jelly extradural clot with much free red blood extruded through the bony opening under high pressure and upon enlarging this opening, a large amount of blood-clot of almost 6 ounces was removed, permitting the underlying depressed dura to rise and thereby relieving the compression of the underlying cerebral cortex. The middle meningeal artery was ligated by a silver clip to prevent more bleeding. The dura itself was of normal appearance and pulsated normally, and therefore it was not considered advisable to open it. After all the extradural blood-clot had been removed, the usual closure was made with 2 drains of rubber tissue inserted down to the dura. It was now noted that the hematoma, which was situated anterior to the operative incision, had become much smaller and less tense due undoubtedly to much of its bloody contents having been removed when the extradural hemorrhage was evacuated during the operation—its blood having returned intracranially through the underlying line of fracture. Duration, 50 minutes.

Post-operative Notes.—An uneventful recovery; the weakness of the right arm and of the right side of the face entirely disappeared within 6 hours after the operation and the suggestive Babinski also could not be obtained at that time. The general condition of the child immediately improved; the wound healed *per primam*—all skin sutures having been removed upon the fifth day after operation.

Examination at discharge (14 days after operation).—Temperature 98.8° ; pulse, 80; respiration, 24; blood-pressure, 112. No complaints except the soreness over the left side of the head. The operative incision has healed perfectly; no protrusion but a normal pulsation is visible. The hematoma has entirely disappeared. No paralysis of the right side of the face or right arm ascertained; no sensory impairment nor astereognosis. Pupils equal and react normally. Reflexes active but otherwise negative. Fundi—retinal veins possibly enlarged; all details of both optic disks clear and distinct.

Last Examination (May 16, 1919—9 months after injury).—No complaints; patient has done well in school since January—the third month after the injury. Operative area depressed; slight pulsation palpable. No weakness or awkwardness of the right side of the body; no speech impairment. Reflexes active but otherwise negative. Fundi negative.

Remarks.—It is possible that repeated aspirations of the hematoma overlying the site of the fracture of the vault and the extradural hemorrhage could have drained this extradural hemorrhage successfully, if the blood had not gradually coagulated and formed an extradural clot; possibly more frequent aspirations of the hematoma would have lessened and delayed the formation of this clot, and yet the bleeding from the left middle meningeal artery was so profuse that it is very doubtful whether this method of treatment would have been eventually satisfactory. The clear cerebrospinal fluid under an increasing pressure and the presence of a fracture of the left vault in the vicinity of the left middle meningeal artery, together with the localizing signs of weakness of the right arm and of the right side of the face and the other neurological signs, made the diagnosis of an extradural hemorrhage a most probable one. The absence of aphasia or paraphasia is interesting, especially in a patient who is right-handed and whose ancestors and relatives are all right-handed; how the motor speech area escaped being compressed sufficiently to cause an impairment of speech is difficult to conceive, especially since the extradural hemorrhage lay directly over the posterior portion of the third left frontal convolution. It is possible that an aphasic impairment would have occurred if the operation had been delayed several hours longer.

The rapid subsidence of the signs of the intracranial lesion and of the cerebral impairment was most impressive following the operation and the excellent recovery of normality of this patient is gratifying, as it was feared at the time of the operation that too long a delay had been permitted and that an earlier operation would have been more advisable.

CASE 161.—Acute severe brain injury associated with high intracranial pressure due to supracortical hemorrhage and with no fracture of the vault of the skull ascertained. Bilateral decompression and drainage. Excellent recovery.

No. 881.—Murray. Ten years. White. School. U. S.

Admitted August 13, 1917. Polyclinic Hospital.

Operation August 17, 1917—4 days after admission. Bilateral decompression and drainage.

Discharged September 14, 1917—27 days after operation.

Family history negative.

Personal history negative.

Present Illness.—While crossing the street, patient was knocked down by an automobile; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (one hour after injury).—Temperature, 98°; pulse, 132; respiration, 32; blood-pressure, 104. Unconscious and in severe shock. Small hematoma over the vertex of the vault. No bleeding from nose, mouth or ears; no orbital nor mastoid ecchymoses. No paralyses ascertained. Pupils widely dilated and do not react to light. Reflexes all abolished. Fundi negative.

Treatment.—Vigorous shock measures instituted, especially heated blankets, hot water bottles and rectal enemata of hot black coffee; absolute quiet—no further examination being made until patient should recover from this extreme condition of shock. After 48 hours, patient gradually emerged from the shock.

Examination (60 hours after admission).—Temperature, 99.2°; pulse, 90; respiration, 22; blood-pressure, 114. Semiconscious, but cannot be aroused sufficiently to answer questions. Left orbital but no mastoid ecchymosis; otoscopic examination negative. Bimanual examination of the vault negative. No paralysis elicited. Pupils of moderate size, equal, but react to light sluggishly. Reflexes—patellar exaggerated and equal; no ankle clonus but suggestive double Babinski; abdominal reflexes absent. Lumbar puncture—bloody cerebrospinal under increased pressure (20 mm.). X-ray (Doctor G. W. Welton)—"no fracture visible."

Treatment.—Expectant palliative treatment continued for 24 hours longer, but as the signs of an increasing intracranial pressure progressed so that a double papilledema of 1 diopter was revealed in the fundi and a second lumbar puncture registered a pressure of 24 mm. and the pulse had also descended to 70, therefore an immediate right subtemporal decompression was performed.

Operation (4 days after admission).—Right subtemporal decompression (primary anesthesia): usual vertical incision, bone removed, and no complications. Dura was very tense and somewhat bluish, and upon incising it bloody cerebrospinal fluid spurted a distance of 3 feet, revealing a very edematous cortex under very high pressure so that it protruded and ruptured to a depth of 1 cm.; no pulsation visible. An attempt to tap the right ventricle was unsuccessful. No evidence of any large blood-clot, but se
punctate hemorrhages were present in the underlying cortex which ha
appearance of being "water-logged." Much difficulty encounter
attempting to close the incision owing to the bulging of the cerebral
which did not pulsate. An immediate left subtemporal decompressio
considered advisable. Usual closure with 2 drains of rubber tissue in

Left subtemporal decompression: usual vertical incision, bone re and no complications. Dura was bluish and very tense, and upon incising it much bloody cerebrospinal fluid welled out, allowing the swollen cortex to bulge and then to pulsate for the first time. Cortex was in good condition, except in lower part of the operative field where it appeared to have been contused. No evidence of gross blood-clot was found: occasional punctate hemorrhage in the cortex was visible. At the end of the operation, the brain pulsated almost normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 90 minutes.

Post-operative Notes.—Child had a very stormy convalescence; semiconscious and irrational for almost one month, requiring an unusual amount of sedatives; temperature remained about 102°. Left eye "cleared up" one week after the operation, while the right eye continued to be blurred by edema one week longer. The general condition of the patient gradually improved, he became less and less irrational after the signs of an increased intracranial pressure subsided, so that he could be promptly discharged. A photograph was taken on the eighth day post-operative; the left orbital ecchymosis is still visible (Fig. 179).

Examination at discharge (27 days after operation).—Temperature, 98.2°; pulse, 80; respiration, 24; blood-pressure, 116. Perfectly conscious; apparently retarded slightly in thought and expression, but parents say "he was always that way." Occasional dull headache; otherwise feels well. Both operative areas flush with the surrounding scalp; normal pulsation. Hearing negative. Pupils equal and of normal reaction. Reflexes active but equal. Fundi—retinal veins enlarged; lower nasal quadrant especially of right optic disk, slightly obscured by edema.

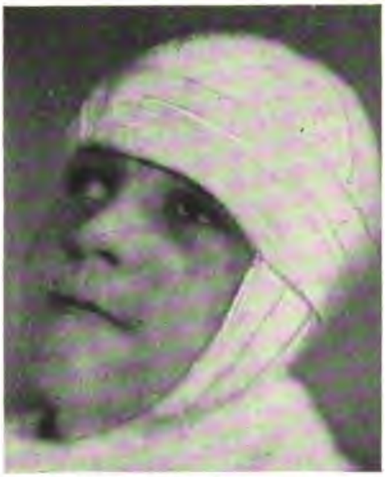


FIG. 179.—The eighth day post-operative of a patient having an extreme intracranial pressure due to a large supracortical hemorrhage, and not associated with a fracture of the skull; bilateral decompression and drainage. Excellent recovery.

Treatment.—Parents cautioned to keep the boy from all active mental and physical exercise; not to attend school for one year.

Examination (September 6, 1918—13 months after injury).—No complaints except an occasional headache of mild severity. Decompression areas slightly depressed, normal pulsation. Reflexes active but otherwise negative. Fundi—retinal veins possibly larger than normal; no blurring of the details of the optic disks. Patient is permitted to begin school next week but must not study much; if the headaches should occur, then he must remain away from school another year.

Last Examination (April 20, 1919—19 months after injury).—No complaints; teacher states, "Murray can do his work as well as the other boys." He is possibly more unstable emotionally than formerly. Operative sites depressed; pulsation not so evident. Reflexes active but otherwise negative. Fundi—retinal veins of normal size; details of the optic disks clear.

Remarks.—The necessity of performing a bilateral decompression in children having brain injuries is much less frequent than in adults, although in these latter patients it is not over 5 per cent. of the operated patients that the intracranial pressure is so high that one decompression alone will not suffice to relieve the pressure satisfactorily; in children, the percentage of these patients requiring a bilateral decompression is not over 3 per cent.—at least, this is the percentage in this series of patients. Children are able to resist more successfully by adaptation a higher increased intracranial pressure than is possible for adults, and they also can absorb by natural means a larger amount of intracranial hemorrhage and edema, so that if the operation of decompression and drainage is necessary, usually one operation alone suffices.

A longer period of time must elapse in this case before it can be stated that the end-result is an excellent one; it is difficult to conceive that this patient will not be impaired in some way—mentally and especially emotionally, and also there is the great danger of epileptiform seizures should dissipation in any form occur later, particularly alcoholism.

It is fortunate in this patient that a right subtemporal decompression was performed first, since the rupture of the underlying cortex might have permanently injured the motor speech centre if the operation had been performed on the left side first. In patients having high intracranial pressure, it is better judgment always to operate upon the side opposite to the supposed speech centre, unless the signs indicate a local lesion in this area; in selected cases of very high intracranial pressure, the first operation should be performed on the opposite side (the safer procedure), and then the second operation over the site of the lesion.

E. *Acute severe brain injuries associated with varying degrees of intracranial lesions. Death. Autopsy.*

CASE 162.—Acute severe brain injury not associated with a fracture of the skull, but with severe initial shock and no intracranial hemorrhage—merely a "wet," edematous condition of the brain; no signs of an increased intracranial pressure. No operation. Death from shock; autopsy.

No. 80.—Veronica. Ten years. White. School. U. S.

Admitted September 24, 1914.—40 minutes after injury. Polyclinic Hospital. Referred by Doctor Alexander Lyle.

Died September 24, 1914—9 hours after admission. Shock.

Family history negative.

Personal history negative.

Present Illness.—While crossing the street, patient was knocked down by an automobile; unconscious for several minutes but became semiconscious en route to the hospital in the automobile.

Examination upon admission (40 minutes after injury).—Temper
97.6°; pulse, 130; respiration, 30; blood-pressure, 90. Semiconsciou
not be aroused to answer questions; very restless. Extensive abrasio
contusions of entire forehead; multiple contusions of head and entir
Slight bleeding from the nose and both ears; no cerebrospinal fluid ob
double orbital but no mastoid ecchymoses. No paralyses ascertained.
slightly enlarged and react to light sluggishly. Reflexes—patellar dif

elicit; no ankle clonus but suggestive right Babinski; abdominal reflexes absent. Fundi negative. No extensive neurological examination made at this time in the hope that with the vigorous treatment of shock, together with the expectant palliative treatment, that the condition of the patient would be improved and then a more thorough examination would be possible.

Treatment.—Vigorous shock measures instituted—external heat by heated blankets and hot water bottles, hot black coffee per rectum, small repeated doses of codeine (grains ½), hypodermically to control the restlessness, absolute quiet, etc. Within 6 hours after admission, the patient did show some signs of improvement in that the temperature became 98.2° and the blood-pressure ascended to 94, but the pulse- and respiration-rates remained over 140 and 38, respectively, becoming weaker and more difficult to auscultate; the patient one hour later suddenly became worse in that the temperature descended to 97.8° and the blood-pressure to 82, and the patient died, 9 hours after admission, from the condition of typical shock following a severe cranial injury.

Autopsy.—No fracture of the skull found. Both tympanic membranes were intact and the source of bleeding from the ears was in several small lacerations of the outer portion of both external auditory canals (illustrating the value of careful otoscopic examinations in all of these patients having a bloody discharge from the ear and yet no cerebrospinal fluid is observed). No intracranial hemorrhage found, but on the contrary the brain was rather pale and anemic and the cortical vessels small. Much cerebrospinal fluid was in the basal fossæ and the cerebral tissues were "wet" and edematous, but not to the extent of being "water-logged" as in an acute cerebral edema. Ventricles were negative.

Remarks.—The signs of this patient as exhibited following her admission to the hospital were all those of severe initial shock following the cranial injury, and during the first 6 hours they were no more severe than frequently occur in many patients who make excellent recoveries. It was thought at the time that this patient would recover from the severity of the shock and especially since the patient was a child, as children can endure the shock following cranial injuries much better than can adults. It would seem, however, that although this patient struggled to overcome the effects of the severe shock, her powers of resistance and especially her vasomotor mechanism were not sufficient to withstand the prolonged effect of the condition of shock.

The treatment of the shock in these patients is most effective in the use of external heat, enemata of hot black coffee and absolute rest and quiet—frequently repeated small doses of morphia in adults and codeine in children hypodermically being most valuable.

The absence of all signs of an increased intracranial pressure, as disclosed by the ophthalmoscope, would undoubtedly have been confirmed, if a lumbar puncture had been performed; no attempt was made to perform a lumbar puncture, however, on account of the severity of the shock and the autopsy findings of cerebral anemia and a general mild edema of the brain would indicate its absence.

It is rather unusual that bleeding from both ears should occur in the same patient and yet there be present an intact tympanic membrane; although there was no cerebrospinal fluid observed, yet that observation does not exclude a fracture of the base of the skull with a resulting laceration of the tympanic membrane. The value of careful otoscopic examinations in all patients having cranial injuries—but after the aural discharge has ceased so that the danger of infection is practically *nil*, is well illustrated in this patient, and no patient should be considered as having a condition of brain injury with a fracture of the skull merely upon the observation that blood was discharged from either ear—and no otoscopic examination made.

Case 163.—Recent severe brain injury following an apparently trivial "bump" upon the head associated with a supracortical hemorrhage and with definite localizing signs. No cranial operation. Death; autopsy; sarcomatosis.

No. 64.—David. Four and a half years. White. U. S.

Admitted July 12, 1914—5 months after cranial injury. Polyclinic Hospital. Referred by Doctor B. Van D. Hedges, Plainfield, N. J.

Died July 14, 1914—8 hours after abdominal operation.

Family history negative; two brothers and one sister well and strong.

Personal history negative; always well and strong; no diseases of childhood.

Present Illness.—Five months ago (February 17, 1914), while playing with his brothers, patient fell upon the ground, striking his head; apparently no loss of consciousness; no bleeding from ears or nose. Upon rising, patient seemed rather drowsy; he was seated in a chair, and one-half hour later, it was noticed that the patient could not move his right leg and within another hour, the right arm became weak and paralyzed; no paralysis of right side of face observed; no aphasia, though a definite slurring of words was noticed. No fever; pulse and respirations were normal; no nausea or vomiting. Within 6 hours, the paralyses became less marked, first in the arm and then in the leg, so that within 36 hours the child was apparently normal. Three weeks later (March 6, 1914), after being in as good health as before the "bump" on the head, the child was observed by the parents to be limping on the right leg, and within 4 hours the right leg was paralyzed and the right arm was much weaker than the left arm; no paralysis of the face. Some drowsiness. No general convulsions, but the fingers of the right hand twitched infrequently. No nausea or vomiting; appetite good; bowels regular daily.

First Examination (March 7, 1914—4 months ago).—Patient examined in consultation with Doctor Hedges and Doctor Robert Abbe. Temperature, 99.4°; pulse, 88; respiration, 24. Well-nourished child; perfectly conscious. Heart and lungs negative. Liver extended ½ inch below costal margin and it was not considered abnormal for a child of 4½ years of age. Spleen just palpable. No abdominal pain or tenderness. The paralysis had alread
sened so that the right arm was almost normal, but the right leg was defi
weaker than the left leg; no facial paralysis. We were unable to induc
child to talk, although the parents said he had been talking normally b
our examination. No disturbance of sensation. No ocular paralyses
than possibly a slight weakness of the left external rectus. No nyst
Reflexes: patellar—right greater than left; no ankle clonus but right

ski was elicited; right abdominal reflexes depressed. Fundi—moderate dilatation of the retinal veins; definite blurring of the nasal margins of the optic disks—left possibly greater than right.

Treatment.—The tentative diagnosis was a small cortical hemorrhage, a possible tuberculoma or a tuberculous meningitis of mild severity; the blood and cerebrospinal fluid by lumbar punctures were advised in order to obtain a Wassermann test and cell count (these were later returned negative). Within 36 hours after this second onset of paralysis, the child gradually became normal, and it remained in its normal good health until July 7, 1914—4 months after the second attack of paralysis; the parents then observed that the child was not moving the right side of its face, and upon examination in my office on July 11, 1914 (4 days later), I found an almost total right facial paralysis (central in origin), and a slight weakness of the right arm; the right leg was apparently normal. Some blurring of speech. No sensory disturbance. Pupils—left smaller than right. No ocular paralysis. No nystagmus. Reflexes: right greater than left; no ankle clonus but a tendency to a right Babinski reflex; abdominal reflexes—right less active than left. Fundi—slight dilatation of the retinal veins, and the blurring along the nasal margins, especially of the left optic disk, still persists. Heart and lungs were negative, but during the routine examination of the abdomen, however, a firm nodular mass—the size of an orange—was palpable in the upper right hypochondrium; it was evidently in the liver, which extended down to the level of the umbilicus; no jaundice was present and apparently no digestive disturbances. The spleen and right kidney were just palpable and apparently normal.

Examination upon admission (July 12, 1914—5 months after the cranial injury).—The neurological examination was the same as at the preceding examination. The laboratory tests of the blood, cerebrospinal fluid, urine and stool, including the tuberculin and luetin tests, were made and were all negative. Doctor J. P. Grant then made an exploratory incision through the right rectus muscle and removed a hard fibrous tumor, the size of a large fist, from the liver; the pathological report was a small-round-celled sarcoma. The child died 8 hours after operation.

Autopsy.—Abdomen: not only sarcomatous masses in the liver, but also extensive involvement of the lymph-nodes of the lesser and greater curvatures of the stomach and in the mediastinum; sarcomatous enlargement of the head of the pancreas and sarcomatous nodules in the right kidney; the adrenals were normal.

Brain: a bluish hemorrhagic clot, 1/8 inch in thickness, was situated in the pia-arachnoid over the precentral area of the left motor cortex, which was very "wet" and edematous, and it extended forward into the left frontal lobe and from the longitudinal fissure downward almost to the left Sylvian fissure; the posterior portion of the third left frontal convolution was partially covered. Careful sectioning of the brain did not reveal any sarcomatous degeneration or any other lesion. No fracture of the vault or of the base of the skull was revealed.

Remarks.—This case is most unusual and the possibilities of diagnosis were many. A "bump on the head"—not worse than is frequently received by children—followed by paralysis, which improves only to return 3 weeks later; another rapid recovery and then a return of paralysis 4 months later. The appearance of a rapidly growing tumor in the liver, which proves at operation to be a small round-celled sarcoma with many metastases, and yet the brain is not involved—only the supracortical hemorrhage of the early "bump" being ascertained with no fracture of the base or of the vault of the skull—which is not uncommon.

The signs of a mild increase of the intracranial pressure associated with a weakness of different portions of the right side of the body and a slight paraphasia (in a child who was right-handed and whose parents were also right-handed), indicated a lesion of the left motor cortex—and a lesion of varying character associated with cortical edema, then convulsive twitchings of the fingers of the right hand pointed to an irritative cortical lesion, and especially in the presence of the ipsolateral pupillary contraction of the left eye. It is surprising, however, that the convulsive twitchings did not persist unless the localized cortical edema about the hemorrhagic area was of such a temporary character that a localized epileptiform seizure was thereby avoided; at the time of the extensive paralysis of the right leg and of the right arm, the localized cerebral edema must have been severe.

If it had not been for the complication of the malignant tumor formation, it would have been possible for the patient alone and without an operation to have "taken care of" the supracortical lesion of hemorrhage and cortical edema, but the natural means of absorptive powers and the general resistance were so lowered by the sarcomatous process that the recovery from the intracranial lesion was not the usual normal one.

CASE 164.—Acute severe cranial injury associated with a compound linear fracture of the right vault; later, definite weakness of the left side of body and with an increased intracranial pressure. Local operation at the site of the laceration of the scalp and the underlying fracture. Brain abscess and meningitis. Death; autopsy.

No. 972.—Rudolph. Five years. White. U. S.

Admitted February 14, 1918—14 days after cranial injury. Coney Island Hospital. Referred by Doctor R. S. Green.

Operation March 8, 1918—24 days after admission. Local operation at the site of compound fracture.

Examination for the first time in consultation—March 16, 1918—45 days after injury.

Died March 18, 1918—10 days after operation and 47 days after injury. Brain abscess and purulent meningitis.

Family history negative.

Personal History.—Two weeks before admission to the Coney Island Hospital, the patient was struck upon the right side of the vault by an iron fragment following a stove explosion in the kitchen of his home; parieto-frontal area lacerated; loss of consciousness for several mi wound sutured by family doctor and patient remained at home in b one week, when increasing headaches and loss of appetite caused the to remove the child to a sanitarium; within the following week, the hea increased in severity and a definite weakness of the left side of th appeared and the child was finally transferred to the Coney Island Hospital, where it was found that the child was stuporous, having a temperature of 100.8°; a definite weakness of the left side of the body was present with increased reflexes upon the left side; otherwise, the patient was in fair condition and remained so until March the eighth (24 days after admission), when the paralysis of the left side became more marked, the temperature ascended to 102.6° and the general condition of the patient appeared to be worse; the laceration over the right fronto-parietal area was enlarged and by means of rongeurs the underlying fracture was widened and explored carefully with a small probe; no signs of infection ascertained; the dura was apparently not torn and was not opened at the operation. Patient, however, progressively became worse until the time of the following consultation:

Consultation (March 16, 1918—45 days after the injury).—Temperature, 104.8°; pulse, 132; respiration, 30; blood-pressure, 108. Profoundly unconscious and moribund. Laceration of scalp apparently healed *per primam*. Neck—slightly stiff and a suggestive positive double Kernig. Complete left hemiplegia and a definite weakness of both the right arm and right leg. Otoscopic examination negative. Pupils—right widely dilated and immobile; left slightly dilated with sluggish reaction to light. Reflexes—all reflexes abolished, both skin and deep reflexes. Fundi—retinal veins are tortuous and dilated; slight edematous blurring of the nasal margins of both optic disks; otherwise negative. Lumbar puncture—turbid cerebrospinal fluid under increased pressure (approximately 14 mm.); "staphylococci."

Treatment.—The child being moribund and the condition one of meningitis most probably of a diffuse character, it was considered advisable merely to continue the expectant treatment and the use of the antimeningitic sera. The condition, however, of the child rapidly became worse and death occurred on March 18, 1918—47 days after the injury.

Autopsy.—Beneath the fracture of the right fronto-parietal bone was a purulent exudate which extended over the entire cortex of the right cerebral hemisphere—particularly its anterior portion. The underlying cortex was rather soft and upon incising it a brain abscess completely filling the entire right cerebral hemisphere was exposed, filled with thick, greenish, creamy pus (staphylococci). Left cerebral cortex negative, except at the base above the middle fossa where there was a small amount of purulent exudate. Line of fracture extended for 4 inches from the middle of the right frontal bone backward into the right parietal bone; the dura had been ruptured beneath one portion of the line of fracture.

Remarks.—This case-history is impressive from the standpoint of the great danger of an infective process, when the dura underlying the fracture of the skull has been torn and especially in the presence of an overlying laceration of the scalp; it is most difficult and at times impossible to prevent an infection of the scalp laceration from occurring—even with the greatest care, but no scalp laceration should be tightly sutured or no drains inserted, and particularly is this true if there is an underlying fracture of the skull.

The gradual development of the tremendous abscess of the entire right cerebral hemisphere, and yet with so few signs of its extent until within several days before the patient's death, is most instructive and merely confirms the belief that there are few intracranial conditions more difficult to diagnose accurately than the condition of subcortical abscess—and especially in the comparatively silent areas of the temporo-sphenoidal and of either frontal lobe, and particularly the right frontal lobe; rarely does the increased intracranial pressure reach a height sufficient to cause a measurable papilledema of the optic disks, and it only does so when the associated meningeal inflammation produces a blockage of the ventricles—and then it is usually too late to afford a definite chance of recovery—operation or no operation.

Case 165.—Acute severe brain injury associated with a compound depressed fracture of the vault and a penetration of the underlying dura. Removal of the depressed fragments of bone. Purulent meningitis. Death; post-mortem examination.

No. 332.—Charles. Eleven years. White. School. U. S.

Admitted July 16, 1915. Fordham Hospital.

Operation July 16, 1915—2 hours after admission. Removal of depressed fragments of bone.

Consultation with Doctor E. R. Cuniffe—July 25, 1915—10 days after the injury.

Died July 26, 1915—10 days after injury. Purulent meningitis.

Family history negative.

Personal history negative.

Present Illness.—While riding in an automobile, patient was thrown out of the car, which had turned over and the iron bar supporting the top penetrated the right occipital area of the skull; immediate loss of consciousness; brought to the Fordham Hospital in the ambulance. The scalp surrounding the wound in the right occipital area was widely shaved, the wound itself cleansed with soap and water and then iodine solution, and by means of forceps the depressed fragments of bone were removed; considerable hemorrhage and a small amount of brain tissue welled through the dural opening; two drains of rubber tissue inserted through dural opening and wet bichloride solution (1–5000) and dressing applied. Patient gradually recovered consciousness and made an excellent recovery; the child felt so well that the parents insisted upon his being removed to his home, which was permitted on July 23 (7 days after injury); the wound had apparently healed *per primam* and merely the small gauze dressing was bandaged over it. Upon arriving home, patient complained of a dull frontal and occipital headache, and 6 hours later a severe general convulsion occurred and the temperature rapidly ascended to 104°; he was immediately brought to the Fordham Hospital again and at a second operation by Doctor Cuniffe, free pus was found in the wound—oozing up through the dural opening; the bony opening
was enlarged by rongeurs, the dural opening widened and two drains o
ber tissue reinserted. The general condition, however, rapidly became

Examination (July 26, 1915—10 days after injury).—Consultatio
Doctor Cuniffe. Temperature, 107.2°; pulse, 164; respiration, 52;
pressure, 106. Well-developed and nourished. Profoundly uncons
weak thready pulse and shallow respiration. Wound dressed, discl
purulent secretion welling up through the dural opening (later bact

cal report—"pneumococci"); rubber tissue drains reinserted and dressing reapplied. Marked stiffness of the neck and bilateral Kernig present. Divergent strabismus—left more than right. Pupils widely dilated and non-reactive to light. Reflexes—patellar absent; no ankle clonus but double Babinski; abdominal reflexes absent. Fundi—retinal veins dilated, tortuous and buried in edematous tissue; double "choked disks" of 4 diopters of swelling. Lumbar puncture—straw-colored cerebrospinal fluid under high pressure (approximately 18 mm.); 15 c.c. slowly removed; later bacteriological report—"pneumococci."

Treatment.—The presence of bacteria in the form of pneumococci in the cerebrospinal fluid at lumbar puncture indicated an extensive diffuse purulent meningitis and as the patient was already in a moribund condition, there was practically nothing therapeutically which could be advised; repeated lumbar punctures every 2 hours with drainage of 10–15 c.c. of the cerebrospinal fluid was advocated, in addition to the routine expectant treatment—the condition, however, was practically hopeless and surely beyond the realm of surgery. The condition progressively became worse—temperature ascended to over 108°, the pulse- and respiration-rates to a point where they could not be counted, and the patient died 18 hours later—on the tenth day after the injury.

Local post-mortem examination: the condition as found at the second operation was confirmed in that the local area explored was saturated in the purulent secretion—undoubtedly an extensive purulent meningitis.

Remarks.—The sudden and rapid onset of the symptoms and signs of a meningitic infection was due chiefly to the site of the infective process being in the occipital area subtentorially, and therefore its extension to the medulla and down into the spinal canal and forward into the basal fossæ of the skull was a most sudden and overwhelming one; the usual preliminary symptoms and signs of headache, increasing stupor and convulsive seizures themselves were thereby absent, until the infective process had become an advanced and extensive one.

In compound depressed fractures of the skull, when the underlying dura has been penetrated, with or without a direct damage to the underlying brain tissue, it is better surgical judgment to perform first, an ipsolateral subtemporal decompression and drainage (even in the absence of a high intracranial pressure), and then the local operation of removing the depressed fragments of bone and the insertion of drains; in this manner, the danger of a localized meningitis is lessened, and if it should occur then the danger of the infected process becoming an extensive and diffuse one is frequently avoided, and it would seem at times even prevented. There is always in these patients an increased intracranial pressure due to the associated edema of the surrounding brain tissue, and if this pressure is lowered by a subtemporal decompression and at times by a suboccipital decompression, then the tissues can resist the infective process much more successfully.

It is unfortunate that a complete autopsy could not be obtained in this case; local examinations are seldom of any real value.

Case 166.—Acute severe brain injury associated with a compound depressed fracture of the vault: removal of the depressed fragment of bone;

cerebral hernia and fungus cerebri. Left subtemporal decompression and repair of hernial protrusion. Meningitis. Death.

No. 244.—Josephine. Eight years. White. School. U. S.

Admitted March 6, 1915. United Hospital, Portchester, N. Y. Referred by Doctor C. H. Bonnell.

Operation March 29, 1915—23 days after injury. Left subtemporal decompression and repair of hernia cerebri.

Died April 5, 1915—5 days after operation. Purulent meningitis.

Family history negative.

Personal history negative.

Present Illness.—While playing in the street about three weeks ago, patient was knocked down by an automobile so that the left side of her head struck a large stone; unconscious for several minutes; carried to the United Hospital, where a fragment of bone of one silver dollar in size was removed by forceps through the scalp laceration over the anterior lower left parietal area of the vault; the underlying dura had been torn so that contused cerebral tissue was lying in the open wound. The intracranial pressure was so high that the dura could not be sutured; two drains of rubber tissue inserted and the scalp laceration was approximated with fine catgut. Child was apparently making an excellent recovery until the bulging of the scalp laceration became more and more pronounced, and on the 14th day after the injury the scalp wound was opened, allowing brain tissue to protrude and then the typical history of cerebral hernia and its resulting fungus followed: the fungoid mass became larger daily and a purulent discharge appeared, while the patient developed a right facial paralysis of the cortical type and a weakness of the right arm associated with a motor aphasia.

First Examination (March 29, 1915—23 days after injury).—Consultation with Doctor Bonnell. Temperature, 103°; pulse, 130; respiration, 32; blood-pressure, 124. Semiconscious; upon being aroused she turns over restlessly and immediately becomes stuporous again. Paralysis of right side of face associated with a definite weakness of the right arm and possibly of the right leg. Unable to talk, although an attempt is made to do so when aroused, but the sound is inarticulate. Over the anterior lower portion of the left parietal bone is an infected fungoid tumor-mass—the size of a lemon; no pulsation visible. Pupils—left larger than right; reacts to light sluggishly. Reflexes: patellar—right greater than left; right ankle clonus (exhaustible) and right Babinski; abdominal reflexes—right absent, left depressed. Fundi—retinal veins full and slightly tortuous; nasal halves of both optic disks blurred—left more than right. Lumbar puncture—slightly cloudy cerebrospinal fluid under increased pressure (approximately 18 mm.); bacteriological report showed no bacteria present—merely numerous leucocytes. X-ray report—"irregular bony opening—4 cm. in diameter—at the site of the hernial protrusion."

Treatment.—In the hope that the hernia and fungus cerebri could be repaired sufficiently to permit a recovery of life at least, a left subtemporal decompression was advised (in spite of the serious and poor condition of the patient); naturally, the danger of a meningitis was very great indeed—

operation or no operation; in fact, the operation would give the patient her only chance of recovery.

Operations (23 days after injury)—*First.* Left subtemporal decompression: usual vertical incision, bone removed, and no complications. Dura very tense, and upon incising it a slightly turbid straw-colored fluid spurted to a height of 6 inches, revealing a very "wet," edematous cortex. Much cerebrospinal fluid escaped, permitting the brain to recede and to pulsate almost normally. No signs of an early localized meningitis observed. Usual closure with 3 drains of rubber tissue inserted. As the fungoid mass was no longer under tension as the result of the ipsolateral decompression, it was thought advisable to attempt its repair. *Second.* Repair of hernia and fungus cerebri: an S-shaped incision of the scalp, both in front and behind the tumor-mass, permitted two large flaps of scalp to be used to cover the cranial defect which was thoroughly cleansed with iodine and then with alcohol; the destroyed and purulent extruded cerebral tissue was now excised, allowing the flap of scalp to be sutured over the deeper portions which receded through the bony opening. No attempt was made to suture the dura since its edges were under the bony rim and they could not be approximated. Three drains of rubber tissue inserted and the usual closure of the scalp made with black silk. Duration, 65 minutes.

Post-operative Notes.—Child in only fair condition; became drowsy, however, within 8 hours and the paresis of the right side of the face and of the right arm improved; speech was not recovered. Three days after the operation, the temperature ascended to 105.6°, the pulse to 138, the neck became rigid and a definite Kernig reflex appeared; a lumbar puncture revealed numerous staphylococci in the cerebrospinal fluid; the condition rapidly became worse and the patient died 5 days after operation and 28 days after injury—a death typical of a diffuse purulent meningitis.

Remarks.—The extreme and late condition of this patient—practically a moribund one—hardly made the operative attempt to obtain a recovery a justifiable one, and yet it offered the patient her only opportunity for recovery. It would have been better surgical judgment to have performed merely the decompression, and then later if the hernial and fungoid mass had improved, then it could have been repaired; the danger of a meningitis was very great in either case—and possibly a localized meningitis was already present at the time of the operation; the absence, however, of bacteria in the spinal cerebrospinal fluid did not conclusively exclude it, except that a diffuse cerebrospinal meningitis was not present.

This case-history is another illustration of the danger of complications in performing a local operation upon depressed fractures of the vault, especially in the presence of an increased intracranial pressure and also in a compound fracture—depression; a preliminary decompression is much the safer and better procedure, since it lessens the danger of a meningitis and also any operative damage to the underlying cerebral cortex at the site of the depressed bone. At times, in selected patients, an ipsolateral decompression may itself suffice together with repeated daily lumbar punctures.

The motor aphasia and right facial paralysis of the cortical type are very characteristic of cortical lesions in this area in right-handed patients.

Case 167.—Acute severe brain injury associated with fractures of the vault and of the base and with extreme cerebral edema, but no intracranial hemorrhage; signs of high intracranial pressure producing an early medullary compression and edema. No operation. Death; autopsy.

No. 993.—George. Five years. White. U. S.

Admitted June 15, 1918 (1.15 P.M.)—15 minutes after injury. Polyclinic Hospital.

Died June 17, 1918 (3 A.M.)—38 hours after injury. Medullary edema.

Family history negative.

Personal history negative.

Present Illness.—Child fell from an open window to the pavement below, a distance of 20 feet, striking upon the forehead; immediate loss of consciousness; carried immediately to the hospital by the mother.

Examination upon admission (15 minutes after injury).—Temperature, 97.8°; pulse, 104; respiration, 26; blood-pressure, 90. Unconscious but very restless; unable to arouse patient sufficiently for him to answer questions. Pulse very weak and irregular. In the middle of the frontal bone was a small punctured scalp wound extending down to the bone; careful probing revealed no depression or fracture of the underlying bone (wound swabbed out with iodine, rubber tissue drain inserted and sterile dressing applied). Profuse bleeding from nose but no cerebrospinal fluid observed. Both orbits swollen and ecchymotic; mastoid areas negative. No paralysis of extremities ascertained. Pupils slightly enlarged but react to light normally. Reflexes—patellar present, left possibly greater than right; no ankle clonus nor Babinski; abdominal reflexes cannot be elicited. Fundi negative. No further examination made at this time in order that the shock might not be prolonged or increased.

Treatment.—Vigorous shock measures instituted together with the routine expectant palliative treatment. Within 6 hours, the general condition of the patient improved in that the temperature ascended to 99°, the blood-pressure to 102, while the pulse- and respiration-rates descended to 96 and 24, respectively; the child became semiconscious but not sufficiently to answer questions; the reflexes became more active but equal and the fundi were negative. It was decided to wait until the following day to perform a lumbar puncture.

Examination (18 hours after admission).—Temperature, 108°; pulse, 80; respiration, 22; blood-pressure, 108. General condition of the patient much better; drowsy and stuporous and yet he answers questions and makes his wants known; complains of "pain in head." Both orbits "swollen shut" and are bluish; right mastoid area ecchymosed. Otoscopic examination negative (bleeding from nose had ceased within 4 hours after admission). Pupils equal and react to light normally. Reflexes—patellar exaggerated but ec
no ankle clonus or Babinski, but no plantar flexion of either foot; abdom
reflexes depressed but equal. Fundi—retinal veins enlarged; lower n
margins of both optic disks slightly blurred by edema. Lumbar punctu
clear cerebrospinal fluid under mild pressure (14 mm.); 10 c.c. slow
moved as a therapeutic means of drainage and this was to be repeated d
an aid to the expectant palliative treatment. X-ray (Doctor G. W. W

—"multiple fractures through the posterior portion of the right frontal bone and the anterior portion of the parietal bone downward toward the base and also a horizontal fracture through the upper portion of the right squamous bone and anteriorly downward toward the right external angular process; no depression of the fragments observed" (Fig. 180).

Treatment.—Expectant palliative treatment continued; careful and frequent observations of the patient as usual. The patient remained in practically the same condition throughout the day until 8 o'clock in the evening (31 hours after the injury), when the child became very restless and confused mentally, attempted to get out of bed three times and had to be restrained in addition to the use of codeine (grains ½, hypodermically) every hour for three doses; the pulse- and respiration-rates were at this time 88 and 26, respectively (due undoubtedly to the emotional excitement and the struggling, and therefore a confusing observation); the temperature had risen to 101.8°; no ophthalmoscopic examination was made at this time. Three and a half hours later (11.30 P.M.), patient became quiet, apparently sleeping; the temperature was 102.4°, the blood-pressure 112, while the pulse- and respiration-rates had descended to 68 and 16, respectively (and considered at this time as being due to the codeine); the fundi, however, disclosed dilated retinal veins with an obscuration of the nasal halves of the temporal margins of both optic disks. No lumbar puncture was performed at this time as it should have been in order to estimate accurately whether the intracranial pressure was rapidly increasing or not. One hour later, the pulse (as taken by the nurse) was only 44 and the respiration but 6 per minute and very irregular—"the child would stop breathing for almost a minute and then begin again rapidly" (typical Cheyne-Stokes respiration); the temperature was 101.4° and the blood-pressure 112; both pupils were contracted and the reflexes were equally increased but no typical Babinski was elicited; the fundi revealed dilated retinal veins and edematous obscuration of all the details of both optic disks—a papilledema, but not one of measurable swelling. An immediate operation was advised, but while the operating-room was being prepared, the pulse- and respiration-rates began to ascend rapidly so that at one o'clock (one-half hour later), they were 78 and 18, respectively, 10 minutes later—94 and 28, respectively, and 20 minutes later—122 and 36, respectively—that is, the sudden onset of an

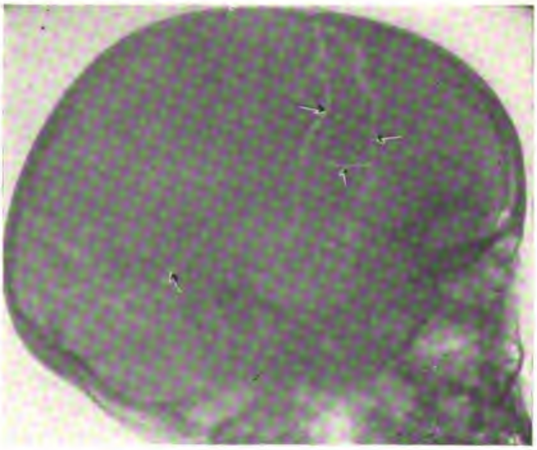


FIG. 180.—Multiple fractures of the entire vault in a patient having an extreme intracranial pressure due to cerebral edema alone—no intradural hemorrhage being present. Early medullary edema and the death of the patient.

acute medullary edema and therefore no operation was performed, as it was realized that all operative procedures were too late and useless. The condition of the patient rapidly became worse in that the temperature quickly ascended to 106°, the pulse- and respiration-rates to 148 plus and 46 plus, respectively, whereas the blood-pressure dropped to 84; pupils became dilated and non-reactive to light, all reflexes were abolished, and the patient died at 3 A.M. (2½ hours after the extreme signs of medullary compression of low pulse- and respiration-rates were observed and 38 hours after injury)—a death typical of acute medullary edema.

Autopsy.—A linear fracture extended from the centre of the right frontal bone obliquely downward and inward along the cribriform plate of the ethmoid bone through the middle of the sphenoid bone, where it almost joined another line of fracture which extended horizontally through the upper portion of the right squamous bone and extending obliquely through the right greater wing of the sphenoid into the body of the sphenoid bone itself; both orbital plates of the frontal bone were fractured and the right anterior portion of the entire skull was slightly movable owing to these fractures extending into the sphenoid bone (Fig. 181). Right temporal muscle was ecchymotic and contained free blood among its fibres, owing to the presence of the fracture of the underlying bone. No hemorrhage intracranially ascertained—either extradural or subdural—but the brain itself, both above and below the tentorium, was very edematous and swollen from the acute cerebral edema—so much so that the cerebral convolutions were flattened; the medulla itself was boggy and "water-logged." Ventricles negative.

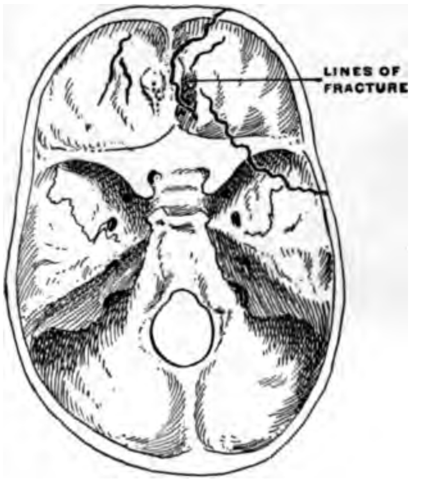


FIG. 181.—Linear basilar fractures of the right middle and both anterior fossæ in a patient having a high intracranial pressure and dying from an acute medullary edema—there being no signs of an intradural hemorrhage.

Remarks.—It was gross carelessness that this patient was not observed more carefully and the signs of an increasing intracranial pressure ascertained earlier by means of the ophthalmoscope and the spinal mercurial manometer, and thus the acute medullary compression would undoubtedly have been recognized and anticipated, so that its early relief by a subtemporal decompression and drainage could have been advised and possibly the recovery of the patient obtained. The patient being a child, however, it was thought that he could withstand and "take care of" any gradually increasing intracranial pressure, and it was not suspected that the pressure intracranially was increasing so rapidly; the fact also that the sudden increase of the intracranial pressure occurred late at night when the patient was under the observation of the nurse alone (the house doctor only to be called following any marked change in the condition of the patient)—this fact also undoubtedly contributed to the unfortunate result of this case.

The absence of an intracranial hemorrhage as a factor in the extreme increase of the intracranial pressure is remarkable only in the fact that these acute cerebral edemas following cranial traumata, with and without a fracture of the skull, occur much more frequently in adults than in children—their most frequent occurrence being in elderly adults having nephritic and arteriosclerotic conditions, and especially in alcoholic adults in whom the typical "wet" brain can be produced by even trivial cranial traumata. Children, however, are usually able to withstand the effects of cerebral edema following cranial trauma, and it is in only rare instances, as in this patient, that the cerebral edema is of such severe degree as to produce an extreme intracranial pressure and its resulting medullary compression, and even medullary edema itself; also, children can withstand the effects of a high intracranial pressure much better than can adults, so that even in the presence of a high intracranial pressure, the signs of a medullary compression rarely appear.

It is surprising that no intracranial hemorrhage, and especially an extradural hemorrhage, occurred following the multiple fractures of the vault and of the base in this patient; no venous sinuses were torn and the right middle meningeal artery escaped rupture. The bleeding from the nose persisted only 4 hours following the injury and no cerebrospinal fluid was observed in it—and this is rather remarkable in that the lines of fracture extended through the ethmoid bones and into the sphenoid bones, but the dura was not torn—an almost impossible escape of the dura following similar fractures in adults.

CASE 168.—Acute cranial injury associated with a fracture of the ethmoid and frontal bones but with no symptoms or signs of a severe intracranial lesion. No operation. Meningitis. Death; autopsy.

No. 301.—James. Five years. White. U. S.

Admitted December 2, 1914—2 days after injury. Polyclinic Hospital. Referred by Doctor T. M. Anderson.

Died December 5, 1914—3 days after admission and 5 days after injury. Purulent meningitis.

Family history negative.

Personal history negative.

Present Illness.—While playing in the street, child was knocked down by an automobile; dazed for several minutes but no loss of consciousness; was brought to the accident room of the hospital in the automobile. Profuse bleeding from nose but no cerebrospinal fluid was observed; contusion of the forehead in the midline with some bogginess and tenderness in this area; nasal bones were found to be fractured. A temporary nasal splint applied and the parents of child were advised to bring him back to the hospital upon the following day. Neurological examination was apparently negative. Two days after injury, mother states the child suddenly complained of severe headache, became drowsy and stuporous and within one hour lost consciousness; an ambulance was summoned immediately.

Examination upon admission (2 days after injury).—Temperature, 102.4°; pulse, 120; respiration, 30; blood-pressure, 112. Unconscious. Both orbits closed by edema and entire frontal area edematous and boggy; clotted blood in both nostrils; no mastoid ecchymoses; otoscopic examination negative. Respiration rather deep and stertorous and very suggestive of Cheyne-Stokes type in its slight irregularity. Occasional twitching of the right facial muscles. No paralyses of the extremities ascertained. Pupils equal and react normally; eyes have a fixed stare. Reflexes—patellar exaggerated, right possibly greater than left; exhaustible double ankle clonus and double Babinski; abdominal reflexes present and equal. Fundi—retinal veins enlarged; entire retinæ edematous and suffused—particularly about the nasal margins of both optic disks, which are obscured by the edema. Lumbar punctures—turbid cerebrospinal fluid under increased pressure (approximately 16 mm.); 14 c.c. slowly removed; bacteriological report (Doctor Jeffries)—"numerous diplococci, most probably pneumococci." Leucocyte count, 27,000; 80 per cent. polynuclears. X-ray (Doctor A. J. Quimby)—"linear fracture of one and a half inches extending upward from the nasal bones into the right frontal bone just to the right of the midline."

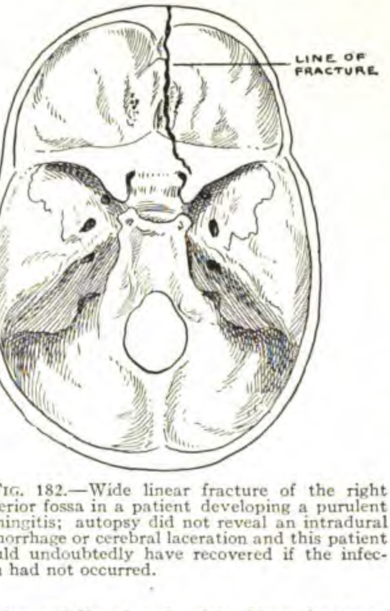


FIG. 182.—Wide linear fracture of the right anterior fossa in a patient developing a purulent meningitis; autopsy did not reveal an intradural hemorrhage or cerebral laceration and this patient would undoubtedly have recovered if the infection had not occurred.

Treatment.—The routine expectant palliative treatment; several doses of the anti-pneumococcic serum were injected; 4 lumbar punctures with removal of 15 c.c. of turbid cerebrospinal fluid were performed within 24 hours. The condition of the patient, however, steadily became worse in that within 30 hours, the temperature had become 104°, the pulse- and respiration-rates 138 and 36, respectively, while the double Kernig test became more marked and the rigidity of the neck caused the position of opisthotonos to be assumed; the respiration became typical of Cheyne-Stokes irregularity; spasmodic twitchings of the muscles occurred all over the body but no general convulsive seizure followed; the patient became profoundly unconscious, the temperature finally ascending to 107.8° when the patient died—66 hours after admission and 105 hours after the injury.

Autopsy (the Coroner's physician refused permission for a post-mortem examination, but after the body had been turned over to the paren
was then obtained by us for the performance of an autopsy).—A f
the nasal bones had extended backward into the ethmoid bones to th
the crista gallæ; the continuation of this line of fracture extende
into the frontal bone just to the right of the median line for a d
almost 2 inches (Fig. 182). The dura overlying the fracture at the
been torn for a distance of one-half inch and it was through this ch

the infective process had travelled. (It is surprising that no cerebrospinal fluid had appeared in the discharge of blood from the nose at the time of the injury, as it would seem from these findings that it must have been present.) Extensive purulent exudate and secretion over the base of the skull, particularly in the anterior and middle fossæ; meningitic exudate extended over both frontal lobes and backward to the Sylvian fissure. No hemorrhage or cortical laceration ascertained. Ventricles also contained a purulent secretion. Infective process had extended down into the spinal canal.

Remarks.—Even if this patient had been kept in the hospital upon being brought there following the cranial injury, it is probable that the infective meningitis would also have occurred in this patient, even with the best of treatment, and yet in all doubtful cases such as this one, the hospital is the proper place for their treatment rather than at home; although the parents denied any irrigations or "cleansing" of the nose itself, yet this method of treatment is so frequent among the laity that its danger and risk are very great indeed. This type of fracture associated with a tear of the overlying dura is a most serious one from the standpoint of a meningitic infection, and a most careful examination of the nasal discharge should always be made in order to ascertain the presence of cerebrospinal fluid or not; if not present, then the risk of an infective meningitic process is slight, whereas if cerebrospinal fluid is definitely found in the nasal discharge, then the patient should be watched most carefully, the expectant palliative treatment rigorously enforced, and if there appear the earliest signs of a meningitic complication—such as slight headache and an increased cell count of cerebrospinal fluid is definitely found in the nasal discharge, then the patient should be watched most carefully, the expectant palliative treatment rigorously enforced, and if there appear the earliest signs of a meningitic complication—such as slight headache and an increased cell count of cerebrospinal fluid, an early subtemporal decompression and drainage should be immediately performed in order to lessen the intracranial pressure and to hasten the cessation of the discharge of the cerebrospinal fluid through the nose (the longer it persists the greater the risk of an infection). In the later cases when bacteria have already appeared in the cerebrospinal fluid at lumbar puncture, these patients are beyond the aid of any surgical treatment, in that the meningitis has been permitted to become a diffuse one and the prognosis of these patients is absolutely bad—no matter what the treatment; the various anti-meningitic sera may be administered in hope, but it is most rare for one of these patients to recover—let alone approximate normality.

CASE 169.—Acute severe brain injury following an apparently trivial "bump" upon the head and causing a linear fracture of the vault; tear of the right middle menigeal artery with the slow formation of a huge extradural hemorrhage. No operation. Death. Autopsy.

No. 271.—William. Seven years. White. School. U. S.

Admitted May 12, 1914—2 days after injury. Polyclinic Hospital. Referred by Doctor C. H. Chetwood.

Died May 12, 1914—40 minutes after admission and 2 days after injury. Acute medullary edema.

Family history negative. *Personal history* negative.

Present Illness.—Two days ago (46 hours), while playing in the school-yard at noontime, the patient struck his forehead upon the brick pavement; no loss of consciousness, but merely stunned momentarily; the school-nurse applied a bandage, made two photographs of the patient (Figs. 183 and 184), and he was able to continue playing after several minutes with apparently only a slightly lacerated wound of the occiput and over the right frontal bone. No bleeding from nose, mouth or ears. The child attended school that afternoon, played after school and that evening complained merely of a slight headache; slept well at night, attended school the following day and except for a slight headache he was able to do his lessons and to play as usual; again complained of the headache at supper that evening and went to bed earlier—immediately after supper. The following morning, child still complained of the headache, but he attended school and during the morning intermission he did not leave his seat and was found there crying by the teacher; "my head hurts." Teacher was assisting him to walk to a rest-room when he suddenly became unconscious. An ambulance was summoned and the patient was immediately brought to the hospital; in the ambulance the patient vomited twice, but did not regain consciousness. No convulsive seizures.

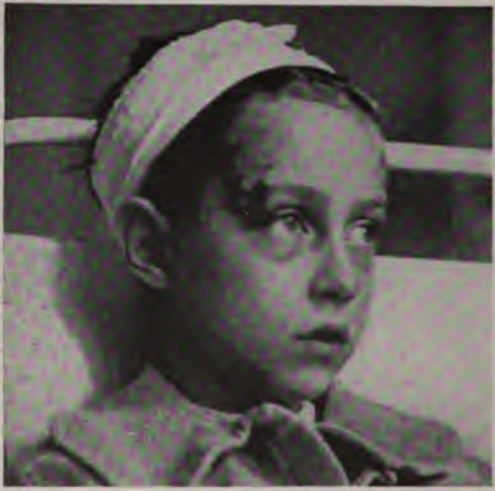


FIG. 183.—Small laceration of the scalp following a "bump" sufficient to fracture the vault and cause a rupture of the right middle meningeal artery.

Examination upon admission (47 hours after injury and 30 minutes following loss of consciousness).—Temperature, 99.8°; pulse, 68; respiration, 14; blood-pressure, 110. Profound unconsciousness. Both eyes were widely opened with conjugate upward deviation. Both pulse- and respiration-rates were irregular and of the Cheyne-Stokes type. Slight contusion and laceration of the scalp of the right forehead and of the occiput. Slight right orbital and right mastoid ecchymoses. Left leg twitched spasmodically. Left side of body possibly more relaxed and limp than right side of body; both arms flexed and spastic while legs were extended and stiff—right more than left. No clotted blood in nares or in any external auditory canals; ostoscopic examination negative. Pupils—right widely dilated while left was contracted to pin-point size. Reflexes: patellar—very much exaggerated, right more than left; double Babinsky, right more than left; double Gordon and Oppenheim present; abdominal reflexes—left absent, right depressed. Fundi—retinal veins tortuous and buried in edematous tissue; double "choked disks" of 2 diopters of swelling and thus all the details of both optic disks obscured as the result of this measurable papilledema. Lumbar puncture—clear cerebrospinal fluid under a high pressure (approximately 26 mm.); only 4 c.c. permitted to escape.

Treatment.—An immediate right subtemporal decompression was advised, and, while waiting for the nearest relative to come to the hospital to give consent for the operation, the patient was prepared by having the head shaved and the operating-room was ordered. Within 20 minutes, however, after the patient's admission to the hospital, the condition of the patient rapidly became worse in that the pulse- and respiration-rates began to ascend rapidly to 84 and 26, respectively, the temperature to 102.8°, while the blood-pressure descended to 100; the left pupil now became dilated and the spasticity of the arms and legs changed to one of flaccidity and an incontinence of the urine occurred. This condition rapidly progressed so that 30 minutes after admission, the temperature had reached 105.6°, the pulse- and respiration-rates 126 and 38, respectively, while the blood-pressure had decreased to 86; the reflexes were all abolished, both pupils widely dilated, and the patient died from a typical medullary edema, 40 minutes after admission.

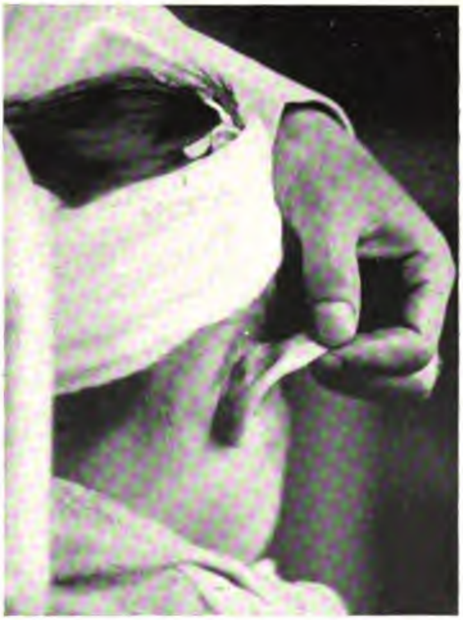


FIG. 184.—Right mastoid ecchymosis in a patient having a supposed trivial "bump" over the right eyebrow. Death occurred two days later from a large extradural hemorrhage.

This patient was within the jurisdiction of the Coroner's office; the Coroner's physician, Doctor T. D. Lehane, after viewing the body in the hospital morgue—in fact, not even touching the body—refused to perform an autopsy in that, "the cause of death is very simple—a fall upon the head, hemorrhage of the brain and death; there is no question as to the cause of death and an autopsy, therefore, is not warranted." In spite of our urgent request and even pleadings to be permitted to ascertain accurately the cause of death and that it might not be so simple as described or supposed and that the clinical history was a most interesting, instructive and even baffling one, yet this omniscient seer could see no reason for permitting an autopsy of the skull to be performed. It is very interesting and an excellent commentary upon such an attitude of a physician of the Coroner's office in that both the father and the mother of the child were not at all satisfied as to the cause of death and, after the Coroner had gone, permission was obtained from them to perform a post-mortem examination of the intracranial contents.

Autopsy.—Linear fracture of 2 inches extended from the posterior portion of right frontal bone obliquely backward into the upper portion of the right *squamous bone*, where it ended (Fig. 185); in its course, it traverse the bone channeled by the right middle meningeal artery and had thus torn it—not all the way through but only a tear of one-third of its wall. A large extradural hemorrhage—the size of a small grape-fruit—compressed the entire right cerebral hemisphere toward the midline and almost one inch beyond the midline and the falx-cerebri was deviated that distance to the left; the right ventricle was completely collapsed and its walls compressed together. Intradurally, there was no hemorrhage or other lesion except the extreme compression due to the right extradural hemorrhagic clot. Subtentorially about the cerebellum and the medulla was much cerebrospinal fluid. No fracture of the base ascertained.

Remarks.—This case-history is an unusual one and yet similar cases occur not so infrequently but that each patient having a cranial injury and followed by persistent headache should be examined at least ophthalmoscopically for fear that a similar intracranial lesion may have occurred. In adults, it is most unusual for a similar lesion to occur and yet not produce the symptoms and signs of its presence much more rapidly and with much greater intensity than in children, in whom, as is well illustrated in this patient, an intracranial lesion of extreme degree may occur within a period of days and yet there be few symptoms and signs of its presence. Careful neurological examinations, and especially the use of the ophthalmoscope and the lumbar puncture needle, are most valuable in their early recognition.

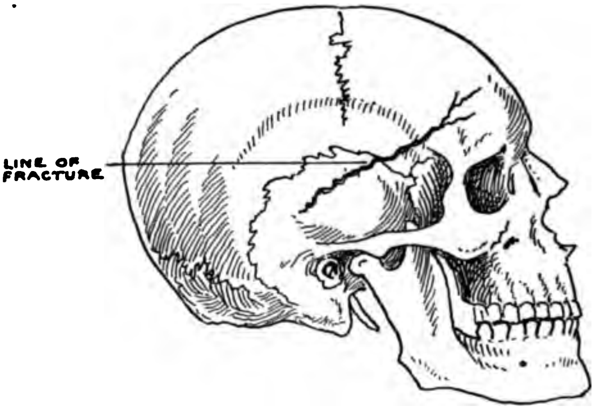


FIG. 185.—Linear fracture of the right vault and of no importance if it had not ruptured the right middle meningeal artery, thereby causing a large extradural hemorrhage to occur slowly and the subsequent death of the patient.

It is rare for an extradural hemorrhage to occur alone in patients having cranial injuries—much more infrequent than the text-books would lead us to believe, and usually these extradural lesions are associated with definite intradural complications of hemorrhages and cerebral edema. The early dilatation of the right pupil and the contraction of the left pupil indicated the paralytic effect upon the homolateral pupil of the compression of the right cerebral hemisphere, while the irritative effect upon the left cerebral hemisphere was revealed in the contracted left pupil; as the intracranial pressure became even higher, then the left pupil, too, became dilated as the patient entered into the stage of medullary edema. The absence of convulsive seizures confirmed the belief that the lesion was an extradural rather than a subdural one.

It is possible that the hemorrhage from the right middle meningeal artery could have been lessened and even prevented to a marked degree, if this child could have been put in bed, an ice-helmet applied, absolute rest and quiet enforced, liquid diet and a daily movement of the bowels assured; codeine, if necessary. In this manner, the blood-pressure could have been definitely lowered and the hemorrhage possibly controlled until the bleeding vessel would have thrombosed at the site of its tear. An excellent recovery could have undoubtedly been obtained in this patient in any event, if the condition had been recognized earlier and before the medullary compression had become extreme and the pulse- and respiration-rates reached their lowest levels; if a right subtemporal decompression had then been performed, it would thus have made possible the evacuation of the extradural hemorrhage and the ligation of the right middle meningeal artery below its point of rupture; after the signs of medullary edema had once appeared in the rapidly rising pulse- and respiration-rates, the ascending temperature and the descending blood-pressure, it was useless to advise any operative procedure as these patients all die—operation or no operation.

Case 170—Acute severe brain injury associated with fractures of the vault and of the base and with a subdural hemorrhage; a mild increase of the intracranial pressure. No operation. Purulent meningitis. Death; autopsy.

No. 274.—Emma. Six years. White. School. U. S.

Admitted May 16, 1914. Polyclinic Hospital.

Died May 28, 1914—12 days after injury. Purulent meningitis.

Family history negative.

Personal history negative.

Present Illness.—While playing tag in the street, the patient was knocked down by an automobile; immediate loss of consciousness; brought to the hospital in the automobile.

Examination upon admission (15 minutes after injury).—Temperature, 97.6°; pulse, 146 plus; respiration, 36 plus; blood-pressure, 90. Unconscious and in extreme shock; pulse scarcely palpable and very thready. Contusion of entire scalp and a boggy hematoma over the right parieto-squamous area—giving the sensation upon palpation of an underlying fracture of the skull. Profuse bleeding from nose and both ears; a large quantity of cerebrospinal fluid mixed in the blood. Left side of body apparently paralyzed—more lax and limp than the right side. Pupils dilated and do not react to light. Reflexes—patellar cannot be elicited; no ankle clonus but an inconstant left Babinski; abdominal reflexes absent. Fundi negative. No further examination of the patient was made at this time for fear of increasing the shock.

Treatment.—Vigorous shock measures—especially heated blankets and hot coffee per rectum, 3 ounces every 2 hours. In spite of the treatment, patient remained in this extreme condition of shock for over 12 hours, and it was only after 24 hours that it could be said that the condition was slightly better; the expectant palliative method was continued and the child gradually recovered from the extreme condition of shock, so that 48 hours after the injury, the temperature had ascended to 99° and the blood-pressure to 104, while the pulse- and respiration-rates had descended to 126 and 30, respectively; a large amount of blood and cerebrospinal fluid continued to discharge from both ears; an ophthalmoscopic examination was practically negative, while a lumbar puncture revealed blood-tinged cerebrospinal fluid and under mild pressure only (approximately 11 mm.). The apparent weakness of the left side of the body had disappeared.

The patient continued to improve for 6 days in that the general condition became more normal and there were no marked signs of an increasing intracranial pressure due undoubtedly to the discharge of straw-colored cerebrospinal fluid from both ears and thus preventing a definite increase of the intracranial pressure; the pulse- and respiration-rates, however, remained above 120 and 28, respectively, and the blood-pressure did not rise above 106; the child remained in a semiconscious condition. Eight days after admission, the temperature suddenly became 105.4°, when the following examination was made:

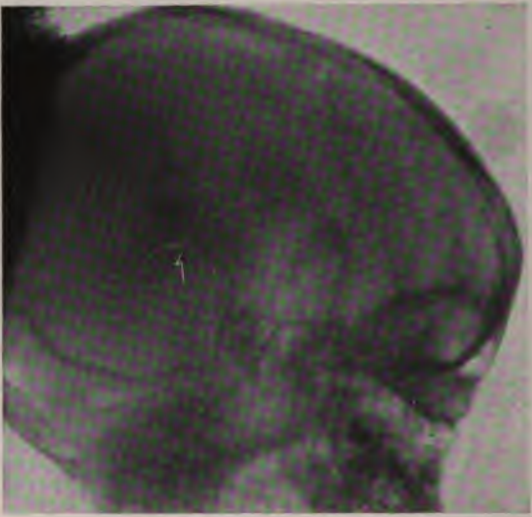


FIG. 186.—Very faint linear fracture of the right squamous bone in a patient dying from a purulent meningitis—the infection having entered through the line of fracture in the right middle ear.

Examination (8 days after admission).—Temperature, 105.4°; pulse, 142; respiration, 34; blood-pressure, 106. Very difficult to arouse and not sufficiently to answer questions. Suggestive rigidity of the neck and a possible double Kernig sign. Discharge from both ears has ceased (3 days before); an otoscopic examination reveals a laceration of the lower halves of both tympanic membranes, and in the right middle ear a small amount of purulent exudate was found. Pupils slightly enlarged and equal. Reflexes—patellar active, left possibly more than right; exhaustible left ankle clonus and suggestive left Babinski; abdominal reflexes difficult to elicit. Fundi—retinal veins enlarged; nasal margins of both optic disks blurred by edema. Lumbar puncture—cloudy cerebrospinal fluid under slightly increased pressure (approximately 11 mm.); bacteriological report—"numerous chains of streptococci observed." X-ray report (Doctor A. J. Quimby)—"curvilinear fracture of right squamous bone extending downward toward right external auditory meatus" (Fig. 186).

Treatment.—The presence of streptococci in the cerebrospinal fluid at lumbar puncture indicated that the meningitis was already a diffuse one and therefore the condition was practically a hopeless one and too far advanced to be benefited in any real way by an operative procedure of drainage. The condition of the patient rapidly became worse—the temperature ascending as high as 108.2°, so that the patient finally died on the twelfth day after injury and the fourth day following the sudden rise of temperature.

Autopsy.—A fracture of skull extended vertically downward through

the right squamous bone into the right petrous bone and transversely across it through the sella turcica and into the left petrous bone and along its posterior crest to the left middle ear (Fig. 187). Both middle ears were filled with a purulent exudate. The middle fossa of the skull was filled with a cloudy purulent exudate which has extended over the surface of both hemispheres. Small hemorrhagic clots lay beneath both frontal lobes, which were slightly contused. No cortical lacerations found. Small amount of purulent exudate subtentorially. Ventricles negative.

Remarks.—It would seem that in this patient, the infective process had extended intracranially through the ruptured tympanic membranes; fortunately no local treatment of the ears had been attempted—such as irrigation, swabbing out with cotton and other such meddlesome procedures which would facilitate the extension of an infective process intracranially. In this patient, it is very probable that the continued discharge of cerebrospinal fluid and blood from both ears for a period of 5 days made it more possible for an infective process to occur in the middle ears and therefore, although this discharge of blood and cerebrospinal fluid lessened and prevented a marked increase of the intracranial pressure, yet it did so at the greater risk of an infection and resulting meningitis; for this reason, the prolonged drainage of blood and cerebrospinal fluid through the ears as a means of lowering and preventing an increased intracranial pressure is a rather dangerous means of drainage, and it is better surgical judgment in these patients, in whom the discharge of blood and cerebrospinal fluid continues longer than 2 days and in whom there are signs of an increased intracranial pressure, that a subtemporal decompression and drainage through a clean area of the scalp is not only a more efficient means of drainage but a much safer one—the risk of the operation being slight compared with the great danger of infection following a prolonged drainage through the ears or nose.

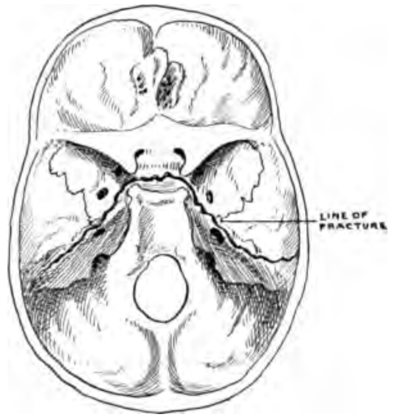


FIG. 187.—Extensive basilar fracture through both petrous bones and across the sella turcica, in a patient developing a purulent meningitis; death twelve days after injury.

It is always surprising in these patients to have the onset of a purulent meningitis appear so suddenly and ushered in by either a rapid increase of the temperature or a convulsive seizure; at times, increasing stupor and headache are very frequent signs and should indicate the necessity of an immediate examination and particularly a lumbar puncture.

The presence of bacteria in the cerebrospinal fluid at lumbar puncture in these patients means that the infective process is a diffuse one and beyond operative treatment; it is only in those cases of localized meningitis where the cerebrospinal fluid at lumbar puncture is free of bacteria—in these patients an immediate decompression and drainage and if necessary a bilateral decompression and drainage is indicated and frequently a recovery of life is obtained. The cerebrospinal fluid may be cloudy due to an increased cell count resulting from the meningeal irritation, and yet no bacteria are present; in these patients also an early operative procedure of decompression and drainage occasionally makes a recovery of life possible.

Case 171.—Acute severe brain injury associated with a fracture of the vault and of the base and with subdural hemorrhage; a gradual increase of the intracranial pressure. Right subtemporal decompression and drainage. Acute purulent meningitis. Death; autopsy.

No. 305.—Henry. Nine years. White. U. S.

Admitted August 1, 1915. Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Operation August 9, 1915—8 days after injury. Right subtemporal decompression and drainage.

Died August 19, 1915—18 days after injury. Purulent meningitis.

Family history negative.

Personal history negative.

Present Illness.—While playing in the street, child was knocked down by an automobile; immediate loss of consciousness; brought to the hospital in the automobile.

Examination upon admission (20 minutes after injury).—Temperature, 97.2°; pulse, 120; respiration, 30; blood-pressure, 98. Profoundly unconscious and in severe shock. Multiple contusions of the head—especially over the left side, and over the entire body. Profuse bleeding from the nose and both ears, with much cerebrospinal fluid in the blood; extensive ecchymoses of both orbits and both mastoid areas. No conjunctival hemorrhage. Pupils dilated and react to light sluggishly. Reflexes: patellar—obtained with difficulty, but apparently equal; no ankle clonus nor Babinski; abdominal reflexes absent. Fundi negative. No further examination made at this time on account of the severity of the shock.

Treatment.—Vigorous shock measures instituted—rectal enemata of hot black coffee, external warmth and absolute rest and quiet. After six hours, patient gradually reacted so that the general condition improved, and at the end of 48 hours the temperature was 99°, the pulse- and respiration-rates were 86 and 26, respectively, while the blood-pressure had risen to 110; the aural discharge had ceased and an otoscopic examination revealed a laceration of the posterior halves of both tympanic membranes; child became semiconscious and it appeared that an uneventful recovery would occur with the expectant palliative treatment alone. The patient, however, did not progress as rapidly as usual and on the sixth day, the ophthalmoscope disclosed the retinal veins enlarged and a definite blurring of the nasal and temporal margins of both optic disks, while a lumbar puncture revealed a blood-tinged cerebrospinal fluid under a markedly increased press
proximately 16 mm.). The condition of the patient gradually
worse in that he became more stuporous and drowsy; the following
tion was now made:

Examination (8 days after admission).—Temperature, 101°;
respiration, 22; blood-pressure, 114. Semiconscious and can only b
with difficulty. Right orbit still closed by edema and the ecch

both orbits and both mastoid areas still persists. Some tenderness over the left posterior parietal area. No Kernig or rigidity of the neck. Pupils equal and react to light normally. Reflexes—patellar very active but equal; no ankle clonus but suggestive double Babinski; abdominal reflexes equally depressed. Fundi—retinal veins dilated; nasal halves of both optic disks obscured by edema. Lumbar puncture—straw-colored cerebrospinal fluid under high pressure (approximately 22 mm.). X-ray report (Doctor A. J. Quimby)—"an irregular linear fracture of lower posterior portion of the left parietal bone, descending into the lambdoidal suture, which is widened" (Fig. 188).

Treatment.—An immediate right subtemporal decompression and drainage advised to lower the increasing intracranial pressure and thus lessen the danger of a later medullary compression.

Operation (8 days after admission).—Right subtemporal decompression (only primary anesthesia required): usual vertical incision, bone removed, and no complications; as there was found free blood in the temporal muscle beneath the temporal fascia, a fracture of the underlying bone was to be expected and a small linear fissure was found only 2 cm. in length; there was a slight depression of the lower fragment but not sufficient for the rongeurs to be inserted and therefore the routine use of the Doyen perforator and burr was made. No extradural hemorrhage ascertained. Dura was tense, bulging and slightly bluish; upon incising it, bloody cerebrospinal fluid spurted to a height of 5 inches and upon enlarging the dural opening much bloody cerebrospinal fluid escaped, revealing a very swollen edematous brain, which began to pulsate after much free blood and cerebrospinal fluid had welled out of the dural opening; no cortical lacerations nor punctate hemorrhages observed. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

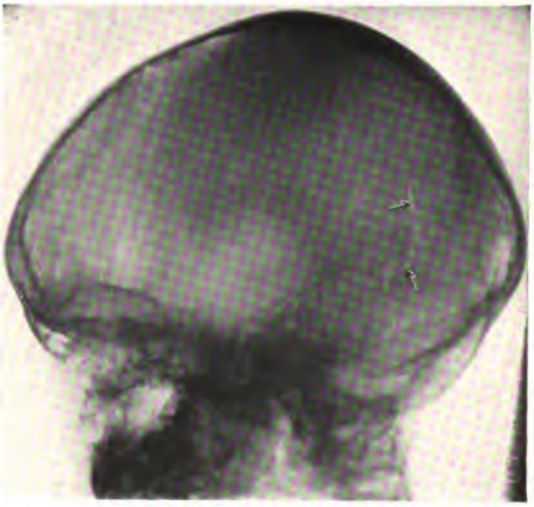


FIG. 188.—Extensive linear fracture of posterior portion of left vault, in a patient dying from a purulent meningitis, most probably resulting from an infective process extending through the lines of fracture involving the base of the skull.

Post-operative Notes.—The operative recovery was uneventful and apparently the child was on the road to an excellent result; the operative incision healed *per primam*, the child became conscious, answered questions and the signs of the high intracranial pressure lessened. While sitting up in bed upon the eighth day post-operative, the patient complained of a severe headache and 4 hours later a general convulsive seizure occurred; the temperature now ascended to 104°, the pulse- and respiration-rates to 118 and 28, respectively, and a profound stupor appeared; a suggestive stiffness of the neck was elicited and also a slight positive Kernig reaction. Lumbar puncture, however, revealed clear cerebrospinal fluid under only an increased pressure of approximately 14 mm. and the cell count was 6 per c.mm. The general condition of the child, however, rapidly became worse, in that the temperature ascended to 107.6°, and the pulse- and respiration-rates to 140 plus and 38 plus, respectively; the patient became moribund and died on the eighteenth day after the injury, the tenth day after operation and the thirty-second hour after the onset of the headache.

Autopsy.—Multiple lines of fracture extended into both orbital plates and ethmoid bones; a line of fracture extended from the site of operation downward into right petrous bone and inward toward the sella turcica, then turned backward across the basilar process, one-half inch behind the posterior clinoid processes, and then forward into the apex of the left petrous bone and along its crest to the left external auditory meatus, and then backward into the left half of the lambdoidal suture which was separated (Fig. 189); the right middle ear contained a small amount of clotted blood, while the left middle ear contained a small amount of purulent exudate (the possible cause for the left earache of which the patient complained to the nurse during his post-operative convalescence and of which we were not aware). The operative site and the contiguous cerebral cortex were of normal appearance and no infective process was present, but over the entire left cerebral cortex, and especially the basilar portions of the inferior surfaces of both frontal lobes and subtentorially, was an extensive purulent exudate—thick, creamy pus. No cortical lacerations or hemorrhages observed. Both ventricles were enlarged from the blockage of their foramina of exit subtentorially.

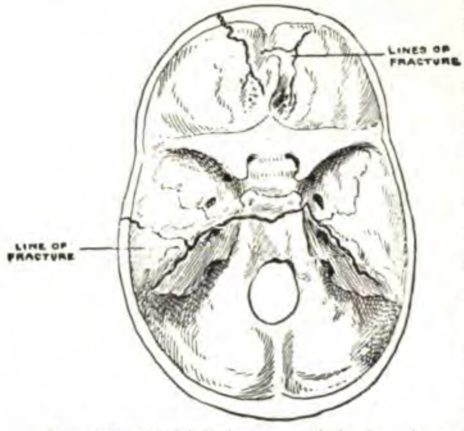


FIG. 189.—Multiple fractures of the base in a patient dying from a purulent meningitis, which resulted from an infective process that had entered intracranially through these channels.

Remarks.—The absence of the purulent meningitis in that portion of the brain and meninges contiguous with the decompression would indicate that the operation itself was not the source of the infection, and the presence of the extensive meningitis in the neighborhood of the lines of fracture extending into the nose and also in the vicinity of the left ear wo
to these fractured lines as being the channels of the infection; the
of a purulent exudate in the left middle ear might have been a seco
rather than the source of the general meningitis, as there had been
lent discharge from the left ear—merely pain which had not be
ered of sufficient importance to notify the house-surgeon. W
operation of decompression, by lessening the pressure of the ad

bral cortex, had permitted these cells to resist successfully the infective process cannot be stated with any certainty, and yet it is a well-known physiological fact that the cells of all body tissues are more resistant to infection when not compressed or under pressure, than they are when under increased pressure. The clear cerebrospinal fluid and the normal cell count, as obtained within 30 hours before the death of this patient, merely indicates that the infective process had not become a diffuse cerebrospinal one and that there was undoubtedly a blockage of the cerebrospinal fluid in the neighborhood of the foramen magnum.

It was rather remarkable to ascertain that the intracranial pressure was increasing 8 days after the injury and yet no extensive intracranial hemorrhage was present—merely a "wet," edematous condition of the brain, and therefore an excellent illustration of a delayed cerebral edema of sufficient height to necessitate its operative lowering; the cerebrospinal fluid was only slightly bloody and blood-tinged, and the prognosis was most favorable, especially in the absence of numerous punctate hemorrhages throughout the cortex. Another interesting observation was the absence of subconjunctival hemorrhages, especially in the presence of such extensive fractures of both orbital plates and a small amount of hemorrhage within the tissues of both orbits. Also the lines of fracture in passing through both petrous bones instead of passing through the sella turcica, as these fractures usually do, they extended posteriorly and then across the basilar process so that at this point the anterior portion of the base of the skull could be rocked upon its posterior portion.

CASE 172.—Acute severe brain injury associated with a linear fracture of the vault and with an increased intracranial pressure. Purulent meningitis resulting from an infected hematoma. Subtemporal decompression and drainage. Death; autopsy.

No. 286.—Josephine. Five and a half months. White. U. S.

Admitted September 10, 1914—3 days after cranial injury. Polyclinic Hospital. Referred by Doctor John A. Bodine.

Operation September 10, 1914—2 hours after admission. Left subtemporal decompression and drainage.

Died September 12, 1914—44 hours after operation. Purulent meningitis.

Family history negative.

Personal history negative; fourth child, normal full-term labor and was considered a normal baby.

Present Illness.—Three days ago while asleep in bed, child was struck over the left side of the head by an older brother with a milk bottle; apparently no loss of consciousness and the child cried, according to the mother, during the entire day; a swelling appeared over the posterior portion of the left parietal area where the overlying scalp was slightly bruised; the child was not considered as being seriously hurt and no more attention was paid to the injury. Thirty-six hours later, however, child became "feverish" and rather drowsy and 6 hours later, definite twitchings of the right side of the body occurred, but no general convulsion. Eight hours later (50 hours after the injury), child became stuporous, no longer noticed anything and the temperature, now taken for the first time, was 104.2° ; it was noticed at this time that the child's neck was stiff. Upon the following day (68 hours after the injury), it was finally thought advisable by the parents to bring the child to the hospital.

Examination upon admission (3 days—70 hours—after injury).—Temperature, 105.6° ; pulse, 138 ; respiration, 36. Well-developed and nourished. Profoundly unconscious; both eyes open and staring vacantly. Position of opisthotonos—the neck and back being very rigid and arched; bilateral positive Kernig—more marked on the left than on the right. Boggy hematoma over posterior portion of the left parietal bone extending over the left half of the occipital bone. No orbital or mastoid ecchymoses. No clotted blood in nose or ears; otoscopic examination negative. Pupils enlarged equally and react to light sluggishly. Reflexes—patellar active, right more than left; no ankle clonus but double Babinski; abdominal reflexes absent. Fundi—retinal veins dilated and tortuous; all details of both optic disks blurred—a papilledema of 1 diopter of swelling. Lumbar puncture—slightly cloudy cerebrospinal fluid under increased pressure (approximately 15 mm.); 10 c.c. carefully removed; bacteriological report (Doctor Jeffries)—"cell count was 40 cells per c.mm. but no bacteria observed."

Treatment.—In the hope that the meningitic irritation and inflammation was not a diffuse one and if a purulent meningitis was present that it was still a localized one, an immediate left subtemporal decompression and drainage was advised in the belief that it offered the patient a definite chance of recovery. (If bacteria had been found in the cerebrospinal fluid at lumbar puncture, then no cranial operation would have been attempted as it would have been recognized that the purulent meningitis was already a diffuse one and beyond the aid of surgery.)

Operation (2 hours after admission and 72 hours after injury).—Left subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications; a transverse line of fracture extended through the upper portion of the left squamous bone and backward below the site of the hematoma; upon inserting forceps along the line of fracture into the hematoma over the posterior portion of the left parietal bone, a purulent exudate welled out (later bacteriological report—"streptococci"). No extradural hemorrhage found. Dura very tense and upon incising it, much straw-colored turbid cerebrospinal fluid escaped and the underlying cerebral cortex was under such high pressure that it bulged and even ruptured before the loss of cerebrospinal fluid and purulent exudate was able to lower markedly the high intradural pressure. No gross hemorrhage or cortical laceration observed. At the end of the operation, the cortex protruded but pulsated slightly and feebly. Usual closure with 2 drains of rubber tissue inserted. Duration, 25 minutes.

Post-operative Notes.—The condition of the child did not imp
18 hours after the operation, the temperature suddenly ascended
the pulse and respiration could not be counted, and this moribund
persisted until the child died, 44 hours after operation.

Autopsy.—Tissues of the scalp of the posterior portion of the
very edematous and boggy; much purulent secretion throughout.

fracture extended from the upper portion of the left half of the occipital bone transversely forward through the lower portion of the left parietal bone and slightly obliquely downward through the upper portion of the left squamous bone forward to the left frontal eminence (Fig. 190); another line of fracture extended downward from this fracture through the anterior portion of the left squamous bone and almost to the middle fossa; no fracture of the base ascertained. The dura underlying the line of fracture beneath the left parietal bone had been torn for a distance of one inch and it was through this channel that the infective process of the overlying hematoma had extended intradurally. Over the entire left cerebral cortex was a purulent exudate and a large collection of pus was found in both the middle fossa and the posterior fossa subtentorially; the cortex of the right hemisphere was less affected, but along the vessels in the sulci the purulent exudate was collected. Ventricles also contained a purulent exudate (bacteriological report—"streptococci"). At the foramen magnum, the medulla had been forced down into it and together with the purulent exudate, the spinal canal was apparently blocked (and this would account possibly for the absence of bacteria in the cerebrospinal fluid at lumbar puncture, 1½ hours before).

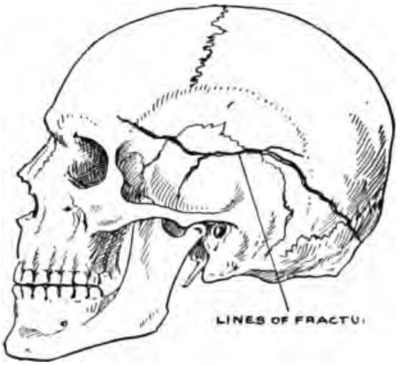


Fig. 190.—Tremendous linear fracture of left vault in a patient dying from a purulent meningitis, due to the extension of infection of an overlying hematoma.

Remarks.—It is most unfortunate that the seriousness of the condition of this child could not have been earlier recognized, so that a mere scalp incision and drainage of the infected hematoma might have successfully prevented the infective process from extending intracranially and if a localized meningitis had already occurred then the local operation, together with a left subtemporal decompression, would have afforded the child a definite recovery of life.

From the findings at autopsy, it would appear that at the time of the operation, the meningitis was already a diffuse one with the exception that the spinal canal had not been invaded by the bacteria and due possibly to their blockage at the foramen magnum as disclosed, and therefore the operation itself was too late to afford the child a real chance of recovery; the absence of bacteria in the cerebrospinal fluid at lumbar puncture made us feel at the time that the operation was advisable—at least, it should be attempted as a definite therapeutic means; if bacteria, however, had been present in the cerebrospinal fluid at lumbar puncture, naturally no cranial operation would have been advisable.

If a meningitis had not occurred in this patient, it is very probable that an excellent recovery of life would have been obtained at home and a good prognosis possible; this case-history merely impresses us again with the great danger of contused and bruised tissues of the scalp and especially associated with an underlying hematoma in the presence of an adjacent fracture of the skull, whether the underlying dura is torn or not—much more so, if the dura is torn. All such hematomata of the scalp having the overlying scalp contused and the X-ray discloses an underlying fracture of the skull—in all of these patients it is much better surgical judgment to incise or aspirate the hematoma through a clean aseptic area of the scalp and thereby lessen the great danger of an infective process occurring in the hematoma and its extension intradurally.

Case 173.—Acute severe brain injury associated with multiple fractures of the vault and of the base and with extradural and subdural hemorrhages and cerebral edema; high intracranial pressure causing the signs of medullary compression and then an early medullary edema. Right subtemporal decompression and drainage. Later infection of hematoma of scalp producing a purulent meningitis. Death; autopsy.

No. 259.—Muriel. Eight years. White. School. U. S.

Admitted April 6, 1914. Polyclinic Hospital. Referred by Doctor W. S. Bainbridge.

Operation April 6, 1914—2 hours after admission. Right subtemporal decompression and drainage.

Died April 18, 1914—12 days after injury. Purulent meningitis.

Family history negative.

Personal history negative.

Present Illness.—While playing upon a fire-escape, child fell a distance of 20 feet, striking the concrete floor upon her head; immediate loss of consciousness; brought to the hospital in the ambulance.

Examination upon admission (50 minutes after injury).—Temperature, 99°; pulse, 60; respiration, 16; blood-pressure, 116. Profoundly unconscious; pulse regular and full and the respirations deep and slightly irregular—suggesting the Cheyne-Stokes type. Extensive hematomata over right frontal and right temporo-parietal areas and over the median portion of the occipital prominence. Profuse bleeding from nose but not from mouth or ears; extensive orbital and right mastoid ecchymoses; right subconjunctival hemorrhage. Otoscopic examination negative. Entire left side of body more limp and relaxed than the right side—undoubtedly paralyzed. Pupils—right dilated, left moderately contracted (one-half hour later, both widely dilated); little or no reaction to light. Reflexes—patellar exaggerated, left greater than right; no ankle clonus but left Babinski; abdominal reflexes absent. Fundi—retinal veins dilated; nasal halves of both optic disks obscured by edema. Lumbar puncture—bloody cerebrospinal fluid under high pressure (approximately 22 mm.).

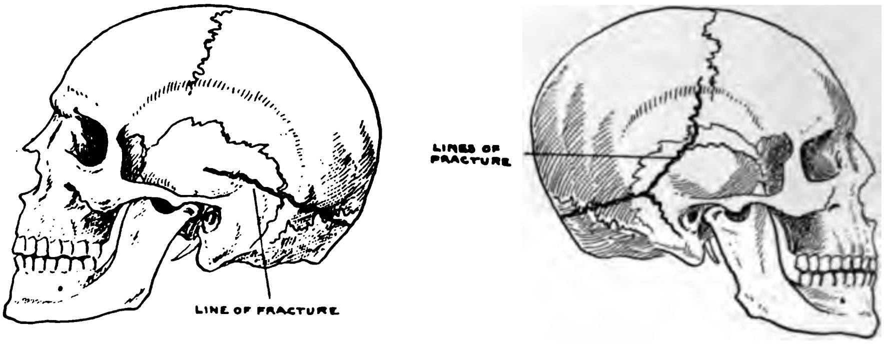
Treatment.—The signs of an acute medullary compression being present and it being feared that the condition of medullary edema might occur, the immediate operation of right subtemporal decompression and drainage was advised; the nearest relatives were summoned, and while waiting one hour for their arrival and the consent for the operation, the condition of the patient rapidly changed in that an incipient medullary edema appeared; within one-half hour, the pulse-rate became 70, the respiration-rate 20, while the blood-pressure descended to 112; both pupils now became dilated and non-reactive to light; one-half hour later, the pulse- and respiration-rates were 80 and 24, respectively, and the blood-pressure 108; one-half hour later (1½ hours after admission), the pulse- and respiration-rates were 100 and 28, respectively, and the blood-pressure 106; one-half hour later, at the time of the operation (2 hours after admission), the pulse- and respiration-rates were 106 and 30, respectively, while the blood-pressure had descended to 104; the neurological examination, however, remained practically the same as upon admission, though the pulse had become rather weak and the respiration shallow and irregular.

Operation (2 hours after admission).—Right subtemporal decompression and drainage: usual vertical incision; much free blood in the tissues of the scalp superficial to the temporal muscle and also in the temporal muscle itself beneath the temporal fascia; a fracture of the underlying bone was therefore expected and three transverse fractures were found extending anteriorly across the lower portion of the right parietal bone and the upper portion of the right squamous bone; the intervening bone fragment was loose and upon removing it, an extensive extradural hemorrhage was exposed and evacuated. Dura tense, bulging and slightly bluish; upon incising it, bloody cerebrospinal fluid spurted to a height of 2 inches, and upon enlarging dural opening much free blood and cerebrospinal fluid escaped but no hemorrhagic clot. The underlying wet edematous cortex tended to protrude but did not rupture owing to the rapid escape of much blood and cerebrospinal fluid. No cortical hemorrhage or laceration observed. Cortex pulsated slightly at end of operation. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—At the end of the operation, the temperature was 102°, pulse 132, respiration 30, and the blood-pressure 104; the general condition was apparently not worse than before the operation. Within 24 hours after operation during which time there had been a profuse bloody discharge from the operative wound, the temperature descended to 102°, the pulse to 108, and the respiration to 24, while the blood-pressure had risen to 112; the child became semiconscious but could be aroused, although unable to speak clearly; recognized mother and understands what she says to her; at the dressing of the wound upon the second day after operation, it was found that the bloody discharge had ceased and although the operative area bulged tensely, yet feeble pulsation was both visible and palpable; the temperature was now only 100.8°, the pulse- and respiration-rates 100 and 24, respectively, while the blood-pressure had increased to 116; the definite weakness of the left side of the body had lessened, although the left reflexes remained increased and the left Babinski persisted; the ophthalmoscopic examination of the fundi disclosed merely an enlargement of the retinal veins and an edematous blurring of the nasal margins of both optic disks. This marked improvement continued until April 11 (5 days after the operation); all sutures had been removed and although the operative area bulged slightly, yet it pulsated normally; at 6 A.M. the temperature was 100°, pulse 94, respiration 24 and the blood-pressure 114; twelve hours later (6 P.M.), the patient had developed a severe headache, definite stiffness of the neck and a double positive Kernig test, while the temperature had suddenly ascended to 105.6°, the pulse- and respiration-rates to 150 and 28, respectively, and the blood-pressure to 118; ophthalmoscopic examination now disclosed dilated retinal veins with a congestion and edematous suffusion of both retinæ; lumbar puncture permitted turbid cerebrospinal fluid to escape under increased pressure (approximately 14 mm.); bacteriological report (Doctor Jeffries)—"many streptococci of the short-chained type."

Treatment.—Four injections of anti-streptococcic serum administered during the next 4 days; repeated lumbar punctures with drainage of 20 c.c. of turbid cerebrospinal fluid were performed—and yet the condition of the patient gradually became worse, so that she died on the twelfth day after the injury—a death typical of purulent meningitis.

Autopsy.—Hematoma over the median portion of the occipital bone was infected—containing much purulent secretion (bacteriological report showed pure streptococci); underlying this infection of the scalp was a transverse fracture of the skull, which had extended from the left squamous bone about 2 cm. above the external auditory meatus backward across the occipital bone and then forward into the right squamous bone, dividing here into 3 smaller lines of fracture, one of which ran obliquely downward into the right mastoid area, while the other two extended forward into the lower portion of the right parietal bone and the upper portion of the right squamous bone and here ended (Figs. 191 and 192). The dura had not been torn by the fracture except in the occipital area just to the right of the median line, and it was by means of this channel that the infective process of the occipital hematoma had extended intradurally and the diffuse meningitis had resulted. The tissues of the right subtemporal decompression were not involved. The posterior fossa subtentorially was filled with the purulent secretion which had extended both forward into the middle fossa and also downward into the spinal canal; the upper portions of the cerebral cortex were not involved. Ventricles contained a small amount of purulent secretion (bacteriological report—"streptococci"). There was no gross hemorrhage or cortical laceration found.



FIGS. 191 AND 192.—Tremendous horizontal linear fracture of the entire posterior vault of the skull in a patient developing a purulent meningitis from an infected hermatoma overlying the line of fracture; death occurred on the twelfth day following the injury.

Remarks.—This case-history is interesting chiefly from 2 standpoints: apparently this is the exceptional and very rare case of severe brain injury producing all of the symptoms and signs of a medullary compression, and then the rapid onset of a medullary edema in the rapidly rising temperature, pulse- and respiration-rates and the quick lowering of the blood-pressure; the operation of subtemporal decompression and drainage is performed in the forlorn hope that an immediate lowering of the high intracranial pressure might make it possible for this patient to recover from the medullary edema—and the patient does make an excellent operative recovery.

The second and the most unfortunate point in this case-history is the rapid development of an acute diffuse purulent meningitis within a period of 12 hours—the apparent onset so rapid and overwhelming that numerous streptococci are found in the cerebrospinal fluid at lumbar puncture as soon as the temperature had suddenly increased to 105.6°, and yet there had been apparently no warning symptoms and signs in order that this most dangerous condition could have been anticipated or at least retarded. The explanation of this lies most probably in the fact that the infective process had entered the posterior fossa subtentorially and had rapidly affected the medulla, extending downward into the spinal canal within several hours and then forward into the middle fossa. In this manner, the cerebral cortex was not involved until late in its progress and therefore there had been few if any warning symptoms and signs. The almost immediate rigidity of the neck and the positive Kernig test are also explained by the early descent of the infective process into the spinal canal.

The importance of draining, or at least, aspirating hematomata which overlie a fracture of the vault, and especially if a tear of the underlying dura is present, is well illustrated by this patient; this is particularly true if the overlying scalp is contused and thus its resistance to infection lowered. If this drainage precaution of the occipital hematoma had been afforded to this patient it is very probable that an excellent recovery, both of life and of normality, would have been obtained.

The pupillary changes in this patient are interesting in that the early dilatation of the right pupil was the paralytic result of high pressure over the right cerebral cortex, and the initial contraction of the left pupil was due to the irritative effect of a lower pressure upon the left cerebral cortex; when this intracranial pressure had so increased that the pressure over the left cerebral cortex had equalled or approximated that over the right cerebral cortex, then the left pupil also became dilated; upon the operative relief of this high intracranial pressure, the pupils returned to their normal size.

Case 174.—Acute severe brain injury associated with a compound fracture of the vault and of the base and with large subdural hemorrhage; signs of extreme intracranial pressure producing medullary compression and the early signs of medullary edema. Bilateral decompression and drainage. Death; autopsy.

No. 487.—Henry. Four years. U. S.

Admitted January 11, 1916. Polyclinic Hospital.

Operations January 11, 1916—1 hour after admission. Bilateral decompression and drainage.

Died September 11, 1916—4 hours after operation. Acute medullary edema.

Family history negative.

Personal history negative.

Present Illness.—While climbing an icy fire-escape, child fell to the ground—a distance of 2 stories; immediate loss of consciousness; patient was immediately carried to the hospital by the mother.

Examination upon admission (15 minutes after injury).—Temperature, 98.4°; pulse, 54; respiration, 14; blood-pressure, 118. Unconscious and in mild degree of shock. Over the right occipital area was a compound fracture of the underlying bone from which macerated cerebral tissue and blood were oozing. Profuse bleeding from mouth and both ears; marked mastoid ecchymoses. (The pulse- and respiration-rates were so irregular and slow and the period of apnœa so prolonged at times—30 seconds and even longer—that a pulmotor was used after attempting artificial respiration for several minutes; the general condition slightly improved.) Pupils dilated and react to light sluggishly. Reflexes all abolished. Fundi—retinal veins enlarged; nasal margins of optic disks obscured by edema. Lumbar puncture—bloody cerebrospinal fluid under high pressure (approximately 20 mm.).

Treatment.—For fear that an early medullary edema would be precipitated by the high intracranial pressure which was already producing the typical signs of a medullary compression, an immediate subtemporal decompression and drainage was considered advisable in the hope that the lowering of this increased intracranial pressure would permit the child to recover. While the operating-room was being prepared, the condition of the child became worse in that the pulse-rate began to ascend and the respiration-rate also, so that by the time it was possible to start the operation the pulse- and respiration-rates were 68 and 18, respectively, while the blood-pressure had descended to 112—the usual signs of an incipient medullary edema following a severe compression of the medulla; it was thought, however, that the immediate lowering of the intracranial pressure might be sufficient to retard and even prevent the progress of the medullary edema, and therefore the operation was performed.

Operations (1 hour after admission).—(No anesthesia being necessary.) *First.* Right subtemporal decompression: usual incision, bone removed, and no complications. Dura very tense, bulging and bluish; upon incising it, dark syrupy blood spurted to a height of at least 3 feet; dural opening quickly enlarged and the swollen hemorrhagic brain protruded through the dural incision—the cortex rupturing in the lower portion of the operative exposure. Much dark free blood welled out of opening, but the hemorrhagic cerebral cortex did not pulsate. An attempt to tap the right lateral ventricle in order to lower the intracerebral tension was not successful. To permit a greater relief of the intradural pressure, a left decompression was now performed. Usual closure with 2 drains of rubber tissue inserted. (It was observed that the right pupil which had been equally dilated with the left now became contracted and thus indicating that the paralytic compression over the right cerebral hemisphere had been changed by the right subtemporal decompression to an irritative one and thus the dilatation of the right pupil became a contraction of it; the left pupil, however, remained dilated as before. Patient regained semiconsciousness for several minutes, but was unable to answer questions; the pulse, however, continued to ascend and was now 76.)

Second. Left subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications; dura tense and bluish and upon incising it, almost pure blood welled out, revealing a tense swollen hemorrhagic cortex which tended to protrude but did not rupture; no cortical laceration or large cortical hemorrhage observed. Much free blood and cerebrospinal fluid escaped so that at the end of this operation the cortex pulsated slightly. Usual closure with 2 drains of rubber tissue inserted. Duration, 1 hour.

Post-operative Notes.—At the end of the operation, the pulse- and respiration-rates had ascended to 84 and 26, respectively, the temperature to 100°, while the blood-pressure had descended to 106; within 2 hours the pulse- and respiration-rates had ascended to 110 and 32, respectively, the temperature to 103.8°, and the blood-pressure had decreased to 98. This condition of acute medullary edema rapidly progressed so that within one-half hour before death (4 hours after operation), the clinical chart was: Temperature, 106.8°; pulse, 144; respiration, 38; blood-pressure, 92—a death typical of medullary edema.

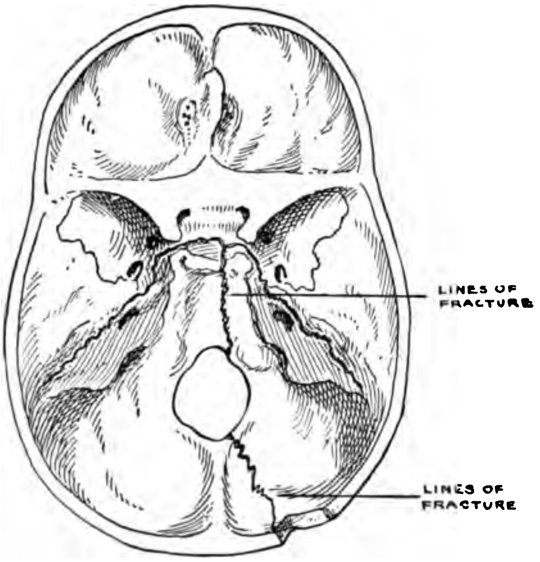


Fig. 193.—Extensive basilar fractures of the occipital bone extending into the foramen magnum and through both petrous bones across the sella turcica in a patient developing an acute medullary compression and an incipient medullary edema within one hour after the injury. A bilateral decompression and drainage failed to permit a recovery of life.

Autopsy.—A depressed fracture of right occipital bone—the depressed area being 2 cm. in diameter; a linear fracture extended downward and forward into the posterior rim of the foramen magnum and then continued forward from the anterior portion of the foramen magnum along the right margin of the basilar process to the sella turcica where the line of fracture divided—one extending into the right petrous bone and the other across the sella turcica into the left petrous bone (Fig. 193). Both tympanic membranes had been lacerated in their posterior halves. The right sigmoid sinus had been torn and, besides a large subtentorial hemorrhage directly compressing the medulla, there was a layer of supracortical hemorrhage due to the tear of several cortical veins as they entered the longitudinal sinus. Multiple punctate hemorrhages were present in the cortex of both cerebral hemispheres, which were greatly swollen from much cerebral edema. The ventricles were negative.

Remarks.—If this patient could have been operated upon one-half hour earlier, while the pulse- and respiration-rates were descending rather than after the pulse- and respiration-rates had reached their lowest level of medullary compression and had begun to ascend, and thus indicating the onset of an acute medullary edema, this patient might have had a slight chance of recovery—a very slight one, however. As it was, he had no chance at all, once the definite signs of an acute medullary edema appeared in the rapidly increasing pulse- and respiration-rates, a rising temperature and a descending blood-pressure; for these reasons, it would have been better surgical judgment to have declined to operate, after it was ascertained that these signs of medullary edema had already appeared while the operating-room was being prepared. It is very difficult in these patients to refuse to operate in the hope that the relief of the high intracranial pressure may permit the patient in occasional cases to recover, and yet these patients do not recover, whether an operation is performed or not, if the definite signs of acute medullary edema have appeared, and therefore no patient in the future should be operated upon in this stage of medullary edema.

The signs of initial shock in this patient were undoubtedly submerged by the rapidly increasing intracranial pressure which produced a very early medullary compression; unless a large intracranial vessel is torn as in this patient, then it is difficult for the signs of an increased intracranial pressure to appear in the presence of severe shock because the lowered general blood-pressure of shock will not be able to cause a large intracranial hemorrhage to occur, as the increasing intracranial pressure will soon be greater than this lowered blood-pressure of shock; in this particular patient, however, the initial shock could not have been severe as the result of the cranial injury and thus the general blood-pressure was not lowered to any marked degree, and so an extensive hemorrhage was possible.

Case 175.—Acute severe brain injury associated with fractures of the vault and of the base and with subdural, cortical and subtentorial hemorrhages; signs of high intracranial pressure and medullary edema. Bilateral decompression and drainage. Death; autopsy.

No. 56.—Henry. Five years. White. U. S.

Admitted September 22, 1913. Muhlenburg Hospital, Plainfield, N. J. Referred by Doctor E. W. Hedges.

Operations September 24, 1913—38 hours after injury. Bilateral decompression and drainage.

Died September 24, 1913—2 hours after operations. Acute medullary edema.

Family history negative.

Personal history negative.

Present Illness.—While running across the road, child was knocked down by an automobile; immediate loss of consciousness; patient was carried to the hospital immediately. Profuse bleeding from nose, mouth and ears, mixed with a small amount of cerebrospinal fluid from each ear; in severe shock in that the temperature was subnormal, while the pulse- and respiration-rates were 140 and 46, respectively. The treatment was the usual expectant palliative one with vigorous shock measures; no return of consciousness.

Examination in consultation with Doctor Hedges (36 hours after admission).—Temperature, 104°; pulse, 154; respiration, 50; blood-pressure, 98. Profoundly unconscious; occasional râles in both lower chests. The pulse was rather irregular and weak and the respirations were shallow. Extensive contusion over the left fronto-temporal area; both orbital and mastoid areas ecchymosed. The bleeding from nose, mouth and ears had ceased; otoscopic examination revealed an extensive laceration of each tympanic membrane. Pupils—slightly enlarged and react to light sluggishly; right internal strabismus. Reflexes—patellar exaggerated, right more than left; no ankle clonus but right Babinski; abdominal reflexes absent. Fundi—retinal veins dilated and tortuous; nasal halves and temporal margins of both optic disks obscured by edema, but no measurable papilledema. Lumbar puncture—bloody cerebrospinal fluid under high pressure (approximately 24 mm.).

Treatment.—On account of the high intracranial pressure, it was thought that an operative lowering of this increased pressure might offer the child a chance of recovery—a mistaken opinion in that it is now recognized that once a patient has entered the condition of acute medullary edema as the result of extreme intracranial pressure, that patient always dies—operation or no operation.

Operations (38 hours after injury).—(No anesthesia being required.) *First.* Right subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications; upon incising dura which was very tense and bluish, almost pure blood spurted to a height of 3 inches and upon enlarging the dural opening, a very hemorrhagic and swollen cortex protruded and almost ruptured from the extreme intradural pressure of supracortical hemorrhage and cerebral edema. As only slight pulsation of the brain was visible at the end of the operation and as the cerebral tension did not become markedly less following the escape of much free subdural blood and cerebrospinal fluid, it was decided to perform an immediate left subtemporal decompression and drainage. Usual closure with 2 drains of rubber tissue inserted.

Second. Left subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications; a transverse linear fracture extended forward along the lower portion of the left parietal bone obliquely downward through the anterior area of the left squamous bone and into the lower posterior portion of the left frontal bone just above the left external angular process; a small amount of extradural hemorrhagic clot removed. Dura tense and bluish; upon incising it, almost pure blood welled out and upon enlarging the dural opening a very tense, swollen hemorrhagic cerebral cortex tended to protrude but did not rupture; much free blood and cerebrospinal fluid escaped permitting the brain to recede slightly and to pulsate feebly. Usual closure with 2 drains of rubber tissue inserted. Duration, 55 minutes.

Post-operative Notes.—Child did not become conscious; the general condition rapidly became weaker in that the signs of medullary edema advanced—the temperature ascending to 106°, the pulse- and respiration-rates to 160 plus and 54 plus, respectively, while the blood-pressure rapidly descended to 86 and lower; severe pulmonary edema occurred and the child died 2 hours after operation—the death of medullary edema.

Autopsy.—Extensive linear fracture of left vault extended from the posterior portion of left parietal bone forward and downward into left frontal bone and then transversely across both orbital plates; another fracture of the base extended transversely through both petrous bones and the sella turcica and into both middle ears (Fig. 194). Multiple punctate hemorrhages throughout the cerebral cortex—more over left cerebral hemisphere and much free subdural blood subtentorially directly compressing the medulla itself. No intracerebral or ventricular hemorrhage.

Remarks.—It was thought that an immediate relief of this high intracranial pressure might afford this patient his only chance of recovery; however, although the patient had survived the severe initial shock in that the pulse- and respiration-rates had at first descended after admission to the hospital and the temperature had risen from subnormal to 100°—undoubtedly the period of medullary compression which was overshadowing the signs of shock—and as the increased intracranial pressure was extreme, the patient rapidly passed into the stage of acute medullary edema without having passed through the stage of a typical medullary compression clinically, in that at no time were the pulse- and respiration-rates below 140 and 40, respectively. From the clinical history and the condition as disclosed at autopsy, this patient would have died—operation or no operation, and no operation should have been performed upon this patient in the belief that it offered him a chance of recovery, for the condition had advanced into the period of acute medullary edema, and these patients in this advanced degree of extreme medullary compression all die—operation or no operation. It is possible that an earlier operative relief of the high intracranial pressure might have prolonged the life of this patient if performed while the pulse- and respiration-rates were descending, and yet no operation would have been advisable upon this patient because the stage of medullary edema appeared before the pulse- and respiration-rates had time to descend below 100 and 30, respectively.

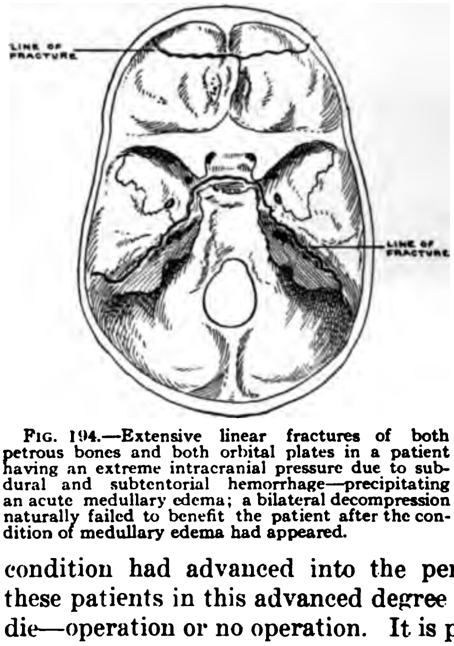


FIG. 194.—Extensive linear fractures of both petrous bones and both orbital plates in a patient having an extreme intracranial pressure due to subdural and subtentorial hemorrhage—precipitating an acute medullary edema; a bilateral decompression naturally failed to benefit the patient after the condition of medullary edema had appeared.

It is rather unusual for the signs of extreme intracranial pressure, as disclosed by the ophthalmoscope and the lumbar puncture needle, to have occurred so quickly following the cranial injury, and they indicated that this extreme intracranial pressure was due to both a profuse hemorrhage and cerebral edema. The autopsy findings of extensive hemorrhage subtentorially would indicate a possible blockage of the ventricles due to a compression of the aqueduct of Sylvius by hemorrhage or edema in addition to the direct medullary compression. It possibly would have been better surgical judgment if the ventricles had been tapped at the first decompression and any blocked cerebrospinal fluid withdrawn and thereby a lowering of the cerebral tension obtained. It must be remembered, however, that no operation should have been advised upon this patient on account of his general condition, and that this patient was in one of the two periods when no patient should be operated upon—that is, the period of acute medullary edema; the other period in these cases when no operation should be performed is that of severe initial shock, when the pulse-rate is over 120, and even 110.

The extensive fractures of the skull, as disclosed at autopsy, indicate the severity of the cranial injury but in no manner lessen the chance of the patient's recovery—rather aided it by affording and facilitating the drainage of the hemorrhage and the excess cerebrospinal fluid of the acute cerebral edema.

CHAPTER XIV

CHRONIC BRAIN INJURIES IN CHILDREN OCCURRING AT THE TIME OF BIRTH

THE CONDITION OF CEREBRAL SPASTIC PARALYSIS IN CHILDREN WITH OR WITHOUT MARKED MENTAL IMPAIRMENT; OBSERVATIONS REGARDING THE OPERATIVE TREATMENT OF SELECTED CASES DUE TO AN INTRACRANIAL HEMORRHAGE AT THE TIME OF BIRTH.

WITHIN the past five years, a distinct advance has been made in the diagnosis and treatment of the condition of cerebral spastic paralysis in children. It can now be ascertained with a high degree of accuracy whether the condition is due to a lack of development of the cerebral cortex and the pyramidal tracts, to a former meningo-encephalitis with resulting destruction of the cortical nerve cells and a thrombosis of the vessels supplying these cells, or to the result and presence of an intracranial hemorrhage at or near the time of birth; cerebral embolus is a less frequent factor, whereas to lues, as demonstrated by the Wassermann examination of both blood and cerebrospinal fluid, including a cell count of the latter and in suspected patients the blood examination of both parents, can only be attributed less than 2 per cent. of the patients (1.7 per cent.). The following observations are based upon the examination of 1922 children up to January 1, 1919, having in varying degrees the condition of cerebral spastic paralysis.[1]

It is rather instructive to trace the recent history of this condition. In 1843, Mr. W. J. Little, of London, in his first monograph upon cerebral spastic paralysis in children or the now so-called Little's disease, and entitled "Deformities of the Human Frame," stated that the condition was due to an impairment of nerve tissues resulting from their lack of development, and also to an earlier meningitis; a few cases following difficult labor were, in his opinion, the result of an intracranial hemorrhage at birth.[2]

It is interesting to note that in his second monograph upon spastic paralysis, published in 1862 (just nineteen years later), and entitled "On the Influence of Abnormal Parturition, Difficult Labors, etc., upon the Mental and Physical Condition of the Child,"[3] he stated that, in his opinion, almost 75 per cent. of these cases were the result of an intracranial hemorrhage. Recent study of this subject with the more accurate methods of modern examination confirms the belief of Mr. Little that about 60 per cent. of these cases of cerebral spastic paralysis occurring in children, with or without marked mental impairment, are due to an intracranial hemorrhage at the time of birth, while the remaining 40 per cent. result from a lack of development of the cerebral cortex or its pyramidal tracts, and also from an earlier meningo-encephalitis following infectious diseases, such as cerebrospinal meningitis, measles, scarlet fever, and whooping cough.

The condition of spastic paralysis in children results most frequently from a lesion of the brain occurring before birth, during birth, or shortly after birth. It is characterized by more or less complete paralysis of the part affected, and is associated with a stiffness or spasticity, depending upon the extent of the involvement of the pyramidal tracts; this hypertonicity produces muscular contractures and deformities, usually flexor in type, with a corresponding overstretching of the opposing muscular groups, usually the extensors. In mild cases, however, the spasticity may be so slight as to cause little or no deformity, but merely an awkwardness of the part affected. Frequently athetoid movements of the arms and legs may be observed, and epileptiform attacks, commonly of the Jacksonian type, may occur. In a large percentage of these patients as the children grow older, not only do the spasticity and its resulting contractures increase, but also their mentality becomes impaired, and this impairment continues until the child may be considered a defective or, still further, an imbecile, and only too frequently an idiot. This mental impairment in the patients having had an intracranial hemorrhage at birth is, in most of the cases, due to the presence of the resulting increase of the intracranial pressure; as the child grows older the mental impairment becomes more and more marked as the result of the long-continued pressure upon the cerebral cells, whereas in the cases due to a simple lack of development, they do not become worse mentally but rather do they improve as the result of training, exercise, etc.

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[1] A preliminary report of this work was made in the *Journal of the American Medical Association*, May 13, 1916.

[2] *The Lancet*, vol. i, p. 350 (December 16, 1843).

[3] Obstetrical Transactions, vol. iii, p. 293 (1862).

One of the most common lesions of the brain producing spastic paralysis is that of intracranial hemorrhage of the newborn. It is of venous origin most frequently, especially the veins overlying the cerebral cortex and the venous tributaries of the longitudinal sinus, and in the more extreme cases even the longitudinal sinus itself may be ruptured; the overlapping of the parietal bones during parturition is the common cause for the injury to the sinus. Naturally, the use of high forceps in difficult labor is an important causative factor in a large number of cases. However, any prolonged difficult labor increasing the venous stasis and partial asphyxia of the child may be sufficient to rupture the delicate vessels overlying the cortex of the brain, and in this way a hemorrhagic clot forms over the surface of the cortex. In some patients the hemorrhage is cortical or subcortical, and therefore, in these cases, direct injury and damage are done to the cortex itself—even a destruction of the cortical nerve-cells and their fibres—whereas in the usual cases in which the hemorrhage occurs *upon* the cortex rather than within the cortex, any cerebral damage is the result of the pressure of the overlying clot and not a primary destruction of the cortex itself; *i.e.*, if it were not for the pressure of the overlying hemorrhagic clot, the cortex would not be damaged at all, and its nerve-cells would be able to function normally; on the other hand, if the hemorrhage is in the cortex or is subcortical, then a real destruction of cerebral tissue occurs, and, once destroyed, there is naturally no regeneration.

According to the extent and pressure of this hemorrhagic clot upon the cortical surface do we find clinically the signs of such interference of the pyramidal tracts; if over the upper portion of both motor areas, then both legs are affected, and a spastic paraplegia results; if over the upper two-thirds of both motor tracts, then both the arms and legs are involved and a spastic diplegia results, whereas if the entire motor area of both cortical hemispheres is compressed, then the extreme form of spastic diplegia results —legs, arms, throat, and face all affected; these extreme cases are the most pitiful ones and, fortunately, they rarely live beyond the age of puberty. In the majority of these spastic patients, however, the hemorrhage extends over one cortical hemisphere alone, the other hemisphere remaining unimpaired, so that a spastic paralysis of the leg or arm occurs opposite to the hemisphere affected; if the hemorrhage extends only over the upper portion of the motor area, then a monoplegia of the opposite leg results, and if over the upper two-thirds of the motor area, then a spastic paralysis of both the arm and leg, and if over the entire motor area, then a total spastic hemiplegia of the opposite side of the body occurs. The upper portion of the motor area is usually more compressed than the lower portion because the hemorrhage is here greater, and as the clot extends downward over the cortex it rapidly thins, so that we may have a marked, spastic paralysis of the leg, and yet the arm be but slightly affected—merely an awkwardness, and the face not at all involved. Then, again, absorption of the clot is a most important factor in lessening the extent of the paralysis; in some cases of mild hemorrhage the clot may be entirely absorbed, only a few fibrous strands remaining as evidence of its existence; these are the patients that later may develop epilepsy in its various forms and show other signs of cortical irritability and instability.

In these cases of intracranial hemorrhage, whether in children or in adults following a cranial injury, unless the hemorrhagic clot depresses the motor area of either hemisphere, or interferes with the pyramidal tracts, then there will be no paralysis, and it is possible for large intracranial hemorrhages to occur, and yet there is no resulting paralysis unless the motor tract is involved; that is, the impairment of the special sense and of the mentality may overshadow the paralysis, and may even exist alone. In this manner, it is possible for the impairment of the mentality to be the chief complaint. However, as in adults with intracranial lesions, a definite disturbance of even the more silent areas of the brain tends to increase the deep reflexes of the extremities, and if the motor tracts are still more affected, then a definite spasticity results.

The treatment of the condition has been a most discouraging one—so much so that the diagnosis of Little's disease implied a hopeless condition; these patients have been the bane of treatment not only to the general practitioner, but to the neurologist and to the orthopedist; apparently very little effort was made to differentiate the three causes of the spasticity, in that the condition was believed to be a hopeless one in any event. The treatment has usually consisted in the older patients of institutional care, general hygienic measures, massage, muscle training, and the various method physical and mental training; nerve resections and their modifications deformities had occurred, then their correction by tendon lengthenings the application of braces; some improvement has been obtained in the lac development cases by ductless gland therapy, but those cases due former meningo-encephalitis and frequently associated with convulsions. that large group of cases due to an intracranial hemorrhage—the treat of these patients has been most discouraging, as the spasticity, if lessened, would return within one year.

Naturally in the cases due to a lack of development of the cortex and its pyramidal tracts, and also those cases resulting from an earlier meningo-encephalitis with destruction of cortical nerve-cells, no cranial operation could be of any value—there being a defective development, and even loss and destruction of nerve tissue. After it had been demonstrated conclusively that the condition of microcephalus was due to a lack of development of cerebral tissue rather than to a premature closure of the sutures of the skull, and that naturally any cranial operative procedure upon these patients could be of no possible benefit (as shown by the operations of Lannelongue and others), the idea of a cranial operation as an aid in the treatment of cerebral spastic paralysis was therefore discarded and remained discredited until the last few years. This lack of differentiation of the three main causes of cerebral spastic paralysis permitted those cases due to an intracranial hemorrhage to escape serious attention, so that they, too, were considered as being hopeless conditions; it was believed that when an intracranial hemorrhage did occur at birth as the result of a difficult labor, the hemorrhage caused a primary destruction of brain tissue and therefore no regeneration was possible, so that in these patients, too, no cranial operation could be of any possible benefit.

A number of years ago, there were many theories regarding the cause of cerebral spastic paralysis. These patients were usually grouped among the mentally defective and classified as defectives, imbeciles and idiots, with or without paralysis. Those cases of spastic monoplegia or hemiplegia, without marked signs of mental impairment, were very puzzling. For many years, it was believed in cases of imbeciles with unusually small heads, that their mental impairment and possible spastic paralysis were due to premature closure of the sutures of the skull which prevented the normal development of the brain—that is, the skull was too small for the brain. The truth was not ascertained until later that the skull did not enlarge because the brain itself did not enlarge and develop normally—that is, the size of the cranium is an index of the size of the brain—only quantitatively, however, not qualitatively. Many cranial operations were devised to offset this supposed premature closure of the sutures of the skull, and so allow the brain to develop—as they thought it would. Trephine openings of various sizes were made in the cranial vault in the hope that the brain would have more room to develop; at times the dura was incised, but more frequently this membrane was left intact. There was no selection of patients made—the fact that the cranium was small was considered sufficient cause for the undeveloped brain; whether there was present an increased intracranial pressure or not was not considered nor ascertained before selecting their cases for operation; and yet, it is surprising that a few of their patients did improve slightly, showing that these few cases, at least, must have had an increased intracranial pressure resulting from a hemorrhage, and that even the inadequate operation was sufficient to produce some improvement. Efforts were made to separate the sutures of the skull under the impression that they had united prematurely and this tremendous operation was repeatedly performed with little or no result. As the dura in the extreme cases of spastic paralysis due to intracranial hemorrhage may be fibrosed and thickened three and four times its normal size, so that it becomes inelastic as in adults, it would have been possible to remove the entire vault of the skull, and yet if the dura were not incised, there could be no relief of pressure obtained nor any enlargement of the intradural capacity possible; that is, to obtain any decompressive effect, the dura must always be incised and allowed to remain open; to resuture it necessarily prevents any permanent relief of the pressure.

During the past few years, the treatment has been directed toward an improvement of the results of the central intracranial lesion upon the extremities and it has been a most discouraging field of work. The operations which have been used in the past and are still being used to improve the condition of spastic paralysis, namely, tenotomies, tendon lengthenings, sections of the posterior nerve roots, alcohol injections of peripheral nerves, nerve resections and other operations, are of only temporary benefit, and it is very rare to see a patient in whom the spasticity has not returned in some degree within one year. In all of the patients treated by the operations just mentioned during the past five years in our clinic, the spasticity began to return within one year after the operation.

Tenotomies have been unsatisfactory. Tendon lengthenings alone are satisfactory in only very mild cases. Foerster's operation for sectioning of the posterior nerve roots of the spinal cord is advocated merely to lessen the irritability and the instability of the cortex of the brain by decreasing the number of afferent stimuli reaching the spinal cord, and also to affect the reflex mechanism of the spinal cord; besides being a rather formidable and long operation for a child, the lessening of the spasticity is only temporary, few cases being reported improved longer than one year; our experience with seven patients has been the same. The injection of alcohol into the peripheral nerves (the Allison and Schwab operation) produces immediate paralysis and a temporary relief from spasticity; in our experience of thirty-one patients, however, the spasticity has returned within one year. With nerve resections (Stoeffell's operation), we have had no experience. Besides in these operations, we do not in any way "get at" the primary cause for the spastic paralysis, namely, the lesion of the brain, but they are merely peripheral operations to relieve the spasticity temporarily, in the hope that, before the recurrence of the spasticity, sufficient power will have returned to the opposing muscular groups to re-establish the muscle balance.

Little, if anything, had been accomplished in improving permanently the condition of spastic paralysis, and the following observations are offered in the hope that they may lead to a more satisfactory solution of the treatment of these most pitiful patients. Attention was first centered on the importance of relieving the increased intracranial pressure as a means of lessening spasticity and improving the mentality of selected patients, by a dec pression operation performed by me in June, 1913, at the Nassau Hosp Garden City, Long Island. The patient, referred by Doctor L. B Rog was a first child, nine years of age, who was apparently normal in every v after an easy delivery until the ninth month of age, when he suddenly a series of epileptic attacks; after these convulsive seizures had subsided was observed that there was a total left hemiplegia with exaggerated reflexes; the left arm and left leg became spastic and gradually assumed the flexor contractures so typical in these spastic patients. Three years ago, the patient had another series of convulsions and since that time these convulsive seizures of varying severity have continued almost daily; the mental impairment was moderate. Every method of treatment had practically been given up as useless. Last June, another series of convulsions began and during the four days preceding my examination of the patient, 302 attacks had occurred; the child was in a condition of status epilepticus—one convulsion following another; the almost continuous administration of chloroform was of little value. In addition to the typical left spastic hemiplegia, the patient had at this examination double "choked disks" as revealed by an ophthalmoscopic examination—that is, a high intracranial pressure, the pulse was 54 and the respiration 8, and oxygen was being used (as a last resource). I advised a right subtemporal decompression in the hope that a relief of the increased intracranial pressure might improve the condition of the patient. No anesthetic was necessary—the patient being unconscious; upon incising the dura, which was exceedingly tense, the cerebrospinal fluid spurted to a height of six inches; the cortex was edematous and swollen, and upon enlarging the opening upward, a fibrous mass, apparently the residue of an old cortical hemorrhage, was exposed lying upon the cortex and extending upward beneath the margin of the decompression opening. As the condition of the child was bad, I decided to remove the mass at a later operation. Owing to the mere relief of the intracranial pressure, the child became conscious at the end of the operation and an uneventful recovery occurred—the child leaving the hospital upon the eleventh day post-operative. The striking feature of the case, however, was the gradual lessening of the spasticity and the contractures of the face, arm and leg, and this improvement continued until the child began using the leg freely and the left hand and arm for picking up articles for the first time in its life; there was also a definite mental improvement.

The thought then occurred: Why not perform a cranial decompressive operation in those selected cases of cerebral spastic paralysis due to a possible hemorrhage upon the brain and showing signs of an increased intracranial pressure? The eyes, therefore, of spastic children were examined carefully with an ophthalmoscope for signs of an increased intracranial pressure. It was very surprising to ascertain that of the patients examined—a large number did show mild though distinct signs of an increased intracranial pressure—that is, a dilatation of the retinal veins, and a hazy edematous blurring of the nasal margins of the optic disks; many of them showed even mild signs of old secondary optic atrophy—rather whitish disks and the physiological cups shallow from scar tissue formation. Doctor Benjamin Farrell and I then began to select for operation such patients having these definite signs of increased intracranial pressure from the various orthopedic clinics—especially the extreme cases and the ones who had received the treatment of tenotomies, tendon lengthenings, alcoholic injections, braces, daily massage and exercises—many of them having been patients during a period of years and with little or no permanent improvement.

During the past six years (until January 1, 1919), I have had the opportunity to examine personally 1922 children having the condition of cerebral spastic paralysis of the diplegic, hemiplegic, and rarely of the monoplegic type; their ages varied from two hours to twenty-five years—the average being four years. In addition to the history and physical findings, there were routine examinations of the fundi of the eyes with the ophthalmoscope in every patient, and a measurement of the pressure of the cerebrospinal fluid at lumbar puncture. This latter test is the most accurate method now known for ascertaining the pressure of the cerebrospinal fluid by means of the spinal mercurial manometer; the normal pressure is 5 to 9 mm. of mercury, whereas in the patients having an increased intracranial pressure due to a former intracranial hemorrhage the column of mercury may rise to 20 mm. and higher, and thus the definite increase of the intracranial pressure is determined. The ophthalmoscopic examination of the fundi is a less delicate test of increased intracranial pressure; naturally, the papilledema and "choked disks" of intracranial tumors and internal hydrocephalus[4] are not to be found in these cases of cerebral spastic paralysis due to a former hemorrhage, as the intracranial pressure in these patients is not sufficiently high to produce these extreme results of high pressure, but their milder signs are exhibited in the blurring and edematous obscuration of the optic disk margins, and frequently the entire nasal halves of the disks; the retinal veins are dilated, frequently tortuous, and their walls thickened with fibrous tissue formation.[5] In conditions of myopia, a similar appearance is also frequently found, but if the measurement of the pressure of the cerebrospinal fluid by the spinal mercurial manometer confirms the ophthalmoscopic findings of an increased intracranial pressure, then there can be no doubt of its presence.

Of the 1922 children examined, having the condition of cerebral spastic paralysis, only 368 of them showed the definite signs of an increased intracranial pressure, and therefore only these patients (about 19 per cent.) were the ones diagnosed as being due to an intracranial hemorrhage, while the remaining 1554 patients, or 81 per cent. of the total number of the patients examined, did not show the signs of an increased intracranial pressure and were therefore classified as being due to a lack of development of cerebral and pyramidal tract tissues, a former meningo-encephalitis, or to an intracranial hemorrhage which was not of sufficient size to produce the signs of an increased intracranial pressure in the fundi of the eyes or in the measurement of the cerebrospinal fluid at lumbar puncture; I believe there are many cases of latent intracranial hemorrhage where the absorption of blood is sufficient to cause later very little increase of the intracranial pressure, and therefore the diagnostic methods now used are not sufficiently accurate to detect the existence of a former hemorrhage. It must also be remem that patients having had a meningo-encephalitis and associated wit sistent convulsions frequently show signs of an increased intrac pressure due to the edematous wet condition of the brain—that is, a condition of external hydrocephalus; in these patients, the history is most helpful in differentiating them from the ones due to hemorrhage.

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[4] *American Journal of Medical Sciences*, April, 1917.

[5] Archives of Ophthalmology, No. 4, 1917.

The history of these 1922 patients has been most instructive: Of the 918 children whose physical and mental impairments were diagnosed as being the result of lack of development of cortical or pyramidal tract nerve tissues, only 73 were not premature babies, and only 89 were not born after a number of pregnancies; that is, the impairment of nerve tissue in these patients was due either to insufficient time for its proper development and growth, as in the premature babies of the seventh and eighth month, or to a less active growth of the nerve-cells themselves, as occurs in children following a large number of pregnancies, where the mothers become malnourished and physically less vigorous. Naturally, none of these children showed signs of an increased intracranial pressure. Syphilis has been demonstrated to be a possible active etiological factor in only 31 children (that is, 1.6 per cent.) of the entire number of 1922 patients examined; a Wassermann test both of the blood and cerebrospinal fluid has been made in each child; in doubtful cases, a cell count of the cerebrospinal fluid has also been utilized and also the blood of the parents examined; in four instances the cerebrospinal fluid of the parents was examined, but with negative results.

Of the 608 cases diagnosed as being the result of a former meningitis and meningo-encephalitis, the history of an acute illness associated with high fever was present in each patient except 77; convulsions had occurred in all but 45 of them. In most of them, the child had been apparently normal until the date of the acute illness, whether it was ten days after birth or two or three years; following the sickness, it was noticed that one side of the body could not be used so well as the other, and then gradually a stiffness of the arm and leg occurred; the convulsions persisted in 327 of these patients, and they were all very unstable children emotionally. When the spastic paralysis followed an acute infectious disease, such as measles, whooping cough and scarlet fever, these cases were usually of the hemiplegic type, and undoubtedly many of them were of thrombotic origin; convulsions usually occurred at the height of the fever, but in many patients they did not continue for more than several days to two or three weeks. Those cases following cerebrospinal meningitis were usually of the diplegic and paraplegic types, and in several of them there were mild signs of an increased intracranial pressure, undoubtedly due to a wet, edematous condition of the brain—a mild condition of external hydrocephalus with and without convulsions.

Of the total number of 1922 children examined, only 368 of them were diagnosed as being the result of an intracranial hemorrhage at or near the time of birth; that is, 19 per cent. of the patients having the condition of cerebral spastic paralysis showed definite signs of an increased intracranial pressure, and these are the patients, and only the ones, that can be improved by lessening this increased intracranial pressure as early as possible after the hemorrhage has occurred.

Let me emphasize (for fear of being misunderstood), first, that we are not operating upon the mentally deficient, the constitutionally inferior and idiots in the hope of restoring them to a normal mentality; and secondly, that we are not operating upon microcephalic children in the belief that the brain will develop and become normal by enlarging the cranial capacity; and thirdly, that we are not operating upon cases of spastic paralysis due to a lack of development and malformation of the cortex of the brain and the pyramidal tracts—cases forming at least one-half of the total number of patients having spastic paralysis—the so-called Little's disease, in which a cranial operation will do no good and from the very pathology of the condition a cranial operation can be of no benefit to the patient. On the other hand, we are operating upon those cases of cerebral spastic paralysis giving a history of difficult labor with or without instruments, and upon ophthalmoscopic examination the definite signs of an increased intracranial pressure are to be seen in the fundus of the eye, and confirmed by the measurement of the pressure of the cerebrospinal fluid by means of the spinal mercurial manometer—*i.e.,* only those cases of cerebral spastic paralysis which show definite signs of increased intracranial pressure, whether this condition is associated with impaired mentality (and it very frequently is as the result of prolonged pressure upon the cortex of the brain), or whether the size of the head is unusually large or small; if, in these latter patients, there is an increased intracranial pressure, then that pressure should be relieved in the hope that the spasticity will be lessened, and the mentality be improved. Naturally, the most satisfactory and desirable patients for operation are the ones with no impairment of the mentality, but these patients are rare when there has been a prolonged increase of the intracranial pressure; much more commonly do we find a more normal mentality in the patients having lack of development and malformation of the pyramidal tracts, unless very extensive.

Naturally, the earlier the diagnosis is made after birth and an operation performed to relieve the intracranial pressure, just so much better is the ultimate prognosis; in the newborn infants under ten days of age, not only will repeated lumbar punctures and spinal drainage and if not successful, then the cranial decompression lessen the intracranial pressure directly, but they will afford a means of drainage of the blood, whereas in the older children the cranial operation is performed merely to offset the pressure effects of the former hemorrhage; if possible, its resulting cystic formation or fibrous mass is removed, but this can rarely be accomplished, owing to the great danger of injury to the underlying cortical nerve-cells. I have now performed this operation of cranial decompression and drainage upon 358 children up to January 1, 1919, with a mortality of 36; that is, about 10 per cent. Their ages have ranged from two hours to twenty-five years; fourteen babies were operated upon the first day after birth with a mortality of two (the hemorrhage being subtentorial and of large amount—producing direct pressure upon medulla), while nine of the remaining twelve children are at the present time apparently normal in every way; eight babies operated upon the second day after birth with no mortality, and five third day, also with no mortality. Naturally, this early diagnosis and tion is the ideal time for the best results to be obtained, for at this earl the supracortical blood can be drained away and the cortical nerve-c thus spared from the superimposed pressure of the hemorrhage. The nosis at this early date is easily confirmed by the presence of blood cerebrospinal fluid at lumbar puncture; the fundi of the eyes rarely show signs of pressure at this early date, unless the hemorrhage is very large.

Some points in the history of these 358 operated patients have been most interesting and instructive; only 52 of them were not first children; only 17 were not full-term babies; only 38 were not born with difficulty—instruments being used, and, particularly, high forceps in a large percentage of them; only 76 did not have convulsive twitchings immediately after birth, and in only 49 children was the spasticity noticed before the eighth month after birth. Of these 358 operated patients, 184 were hemiplegic, 41 paraplegic, and 111 diplegic; many of the patients who were operated upon during the first year of this work in 1913 were the extreme types of the condition—derelicts, as it were, and so badly impaired, both mentally and physically, that only a slight improvement, if any, could be expected in the older cases; the average age of the first 65 operated children was six years, and many of them had never walked nor talked. Naturally, cases of this age and of this extreme type can, at best, be only improved; their cortical nerve-cells have become so impaired from the overlying hemorrhage that any marked return of function is very doubtful; and yet, even in some of these older extreme cases, the improvement has been most striking; seven children, who had never walked, and each of them over eight years of age at the time of the operation in 1913, are now walking.

In the younger children, however, the results have been most gratifying, and it is in these patients under three years of age that the greatest amount of improvement can be obtained by an early operative procedure. Not only has there been a lessening of the spasticity of the arms and legs impaired in these patients selected for operation, but there has been a definite amelioration of the mental condition of the patients to such a degree that in many of the older children their coöperation in the carrying out of the after-treatment can be obtained; this is a most important aid in the physical training of the child.

A written permission for autopsy is obtained from the nearest relative of each patient (private or ward) before operation; in this manner, if the patient should die, then not only will the cause of death be ascertained, but the accuracy of the diagnosis and other valuable data for the treatment of future patients; of the 36 patients who died, an examination of the brain was made in each case, and in all of them, with the exception of two patients the diagnosis of an intracranial hemorrhage was confirmed; the autopsy of these two patients was most instructive; the first one did not have an intracranial hemorrhage but a very "wet," edematous brain under high pressure associated with an enlarged thymus, whereas the second patient revealed an edematous condition of the brain with numerous adhesions and a whitish connective-tissue formation in the sulci about the vessels—similar to the results of a former meningitis.

Moving pictures have been taken of a large number of these patients before operation, and then at intervals of six months following the operation; in this manner, the lessened spasticity and the resulting improvement of gait can be accurately demonstrated and recorded.

The Binet mental tests have been used both before and at regular inter vals following the operation; the mental improvement of many of these children has been most impressive.

METHOD OF OPERATIVE PROCEDURE

In those cases of spastic paralysis of the hemiplegic, paraplegic, or diplegic type with a definite history of difficult labor, with or without the use of instruments, and in whom, upon ophthalmoscopic examination, definite signs of increased intracranial pressure are shown in the dilated retinal veins and a blurring and haziness of the optic disks, especially of their nasal halves, and the cerebrospinal fluid at lumbar puncture is under high pressure as measured by the spinal mercurial manometer, then a large right subtemporal decompression is performed to relieve the intracranial pressure. If the intracranial pressure is extremely high and remains high after the operation, a left subtemporal decompression is performed the following week, the operative recovery requiring only a week or ten days.

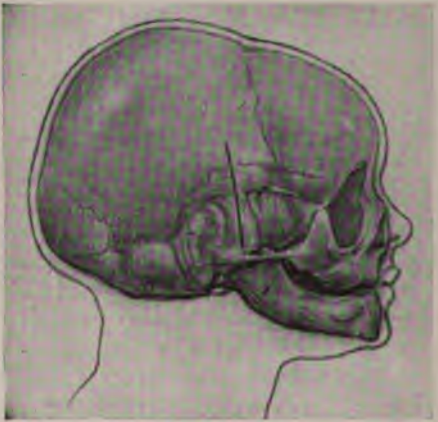

FIG. 195.—Sagittal section of the skull of a baby of three months of age indicating the relations of the vertical incision of a right subtemporal decompression.

The operation itself is a typical subtemporal decompression and consists of a vertical incision over the side of the head, two and one-half to three inches in length, extending from the parietal crest down to a point overlying the zygomatic arch and just anterior to the external auditory meatus—that is, to the lowest level of the middle fossa (Fig. 195). The fibres of the temporal muscle are separated longitudinally, and then a small opening in the squamous bone made by the Doyen perforator and burr is enlarged by rongeurs to a diameter of two to three inches. The dura is now incised in a stellate manner and left open, and by this means of drainage the increased intracranial pressure is relieved permanently. The duration of the operation should not exceed fifty minutes; usually it is only forty minutes. Absolute hemostasis is essential—the less the loss of blood, the less the shock. The loss of a large amount of cerebrospinal fluid should be prevented by elevating the head during the operation, and if the post-operative temperature exceeds 104°, then the head of the bed should be lowered. This operation is not a formidable procedure for one trained in neurological surgery; the anesthetic should be administered by an expert.

The usual pathological findings are definite fibrous or cystic formations resulting from a supracortical hemorrhage occurring at birth. These pathological lesions are treated according to the individual findings—removed, punctured, the outer wall of the cyst excised, and very frequently merely let alone; that is, more damage to the underlying cortical nerve-cells may result by endeavoring to remove the lesion, and therefore it is wiser in many patients not to attempt it. The decompressive operation is performed merely to offset the local effects of the pressure of this hemorrhage with cystic formation and the partial blockage of the excretion of the cerebrospinal fluid by lowering the general intracranial pressure and by draining the excess amount of cerebrospinal fluid, and consequently lessening the spasticity and mental impairment. In 47 patients, the hemorrhagic cyst was cortical and subcortical and naturally in these patients, the nerve-cells and their fibres must have been primarily damaged, so that a marked improvement cannot be expected in them. In 84 patients at operation, the supracortical hemorrhagic cyst was visible, and in all of the operated patients the intradural pressure was abnormally increased.

The dura has been thickened, whitish and fibrous in all of the patients at operation; in not one of these patients was the dura transparent. Even in babies under one year of age, the dura was frequently of a thickness of one-sixteenth of an inch and non-elastic; this fact undoubtedly accounts for the lack of bulging of the fontanelles after the first month following birth in these patients, in that the supracortical and subdural hemorrhage forms a layer of clot over the inner surface of the dura—at times to a thickness of ⅛ of an inch, and then the organization of this blood-clot causes the fibrous thickening of the dura itself due to this connective "scar" tissue formation; not only does the dura become thickened, whitish and non-transparent, but of the greatest importance to the future cerebral development of the child the dura also becomes non-elastic, so that in these patients there is no bulging of the fontanelles and frequently very little pulsation is palpable, and the closure of the fontanelles occurs either at the normal time or frequently earlier than is normal. Ophthalmoscopic examinations and the spinal mercurial manometer will, in these patients, register an increase of the intracranial pressure, whereas the premature closure of the fontanelles and cranial sutures due to conditions of lack of development of the cerebral tissues will naturally not produce any of the signs of an increased intracranial pressure. The appearance of the spasticity being delayed usually until the seventh or eighth month after birth in the hemorrhage cases is most probably due to the progressive effects of this non-elasticity of the dura which prevents the normal development of these children—both physically and mentally, whereas immediately after birth the increased intracranial pressure is compensated, at least temporarily, by the elasticity of the dura making possible the bulging at the fontanelles and the separation of the cranial sutures.

This prolonged increase of the intracranial pressure—months and even years after the original subdural hemorrhage—is rather surprising, and yet the reason for it is obvious. In the mild cases, no doubt there are many latent conditions of intracranial hemorrhage at birth where the blood escaping into the cerebrospinal fluid supracortically is not of sufficient amount to prevent its being absorbed within a few days or weeks at most; that is, similar to many adult patients having brain injuries, with or without a fracture of the skull, in whom a moderate amount of blood from the ruptured intracranial vessels can be absorbed naturally, so that no cranial operation of decompression and drainage is necessary and an uneventful recovery occurs; it should also be remembered in this connection that in some of those so-called latent cases of intracranial hemorrhage where apparently an excellent recovery has been obtained, yet later in life convulsive seizures may occur, and at operation or autopsy there are found numerous supracortical adhesions usually associated with an edematous cortex—an evidence and result of the former supracortical hemorrhage. In the more severe cases, however, of a supracortical hemorrhage at birth, it is of such large amount that it cannot be absorbed normally; then later do we find not only the definite evidence of its presence in the fibrous or cystic formations, in and usually upon the cerebral cortex, but also the walls of the cortical vessels and particularly of the cortical veins and sinuses are very much thickened and fibrosed (just as the dura itself is thickened in the patients having had an earlier supracortical hemorrhage); this thickening of the vessel walls and the overlying dura is the result of the organization of the layer of the supracortical blood, so that the excretion of the cerebrospinal fluid through the stomata of exit in the walls of the cortical veins, sinuses, etc., is thus blocked, and therefore a wet, edematous condition of the cortex results—a mild condition of external hydrocephalus.[6] Just as the condition of hydrocephalus is due in the majority of patients to a former meningitis, and if the aqueduct of Sylvius or the foramina of Majendie or Luschka are blocked by the meningeal exudate or adhesions, then the condition of hydrocephalus interna develops, but if the ventricles are not thus blocked yet the condition of hydrocephalus externa may develop on account of the meningitis being usually a diffuse process, so that it is very probable that the normal stomata of exit for the excretion of the cerebrospinal fluid through the cortical veins and sinuses will have become blocked by the meningeal exudate; in this same manner, a mild external hydrocephalus is not only possible but very liable to result in these conditions of supracortical hemorrhage of the diffuse type occurring at or near the time of birth (Fig. 196). Therefore, the resulting increase of the intracranial pressure in many of these patients having the condition of cerebral spastic paralysis is not only due to the direct increase of the intracranial contents by the escape of blood upon the cortex and into the subdural space with the later fibrous and cystic formations, but also due in very many of the patients to a blockage of the normal excretion of the cerebrospinal fluid through its channels of excretion—the cortical veins and sinuses.

The after-treatment consists of the routine orthopedic treatment which the patients, with the exception of the newborn babies, had all had before operation; the correction of the deformities by tendon lengthenings or merely stretching of the contracted muscles, the maintenance of corrected positions through the employment of specially adapted braces, and skilled massage, particular attention being given to the weakened and overstretche
groups; the usual systematic course in muscle training has been carr
before the operation. Mental training in special schools is most i
Naturally, we do not believe that all patients having the conditi
bral spastic paralysis due to an intracranial hemorrhage
cranial decompression; in some mild cases tendon l

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[6] *American Journal of Medical Sciences*, April, 19

sufficient, and this is especially true in the absence of mental impairment. But those selected cases of spastic paralysis, particularly of the hemiplegic and paraplegic types, which show the definite signs of increased intracranial pressure are the ones that can be very much improved by such a procedure.

In conclusion, I wish to repeat that this is merely a report of the work being carried on to improve the condition of selected cases only of cerebral spastic paralysis in babies and children. Naturally, the earlier after birth the diagnosis of intracranial hemorrhage is made, so that the blood can be drained off in fluid form by a simple operative procedure—either by repeated lumbar punctures of spinal drainage or by a modified cranial decompression and drainage—the better and more normal the child to be obtained; the object of this work is to emphasize the necessity of earlier diagnosis and earlier operations in these selected patients; also the necessity of possibly more careful obstetrics, particularly in the use of high forceps when a Cæsarean section may be preferred. Most of the older children have really been derelicts at the time of operation, and yet, if an improvement can be obtained in these older children, surely a much greater improvement is possible in the younger children. The operation is by no means a cure—except possibly in the newborn infants—and the improvement in our selected patients may be only a temporary one, as sufficient time has not yet elapsed to permit a definite opinion. But from the pathology of these selected cases operated upon and the general continuous improvement which has resulted and is still progressing, I feel justified in making a report of the work in the hope that it may be an aid in the preventive treatment of this very ul condition.

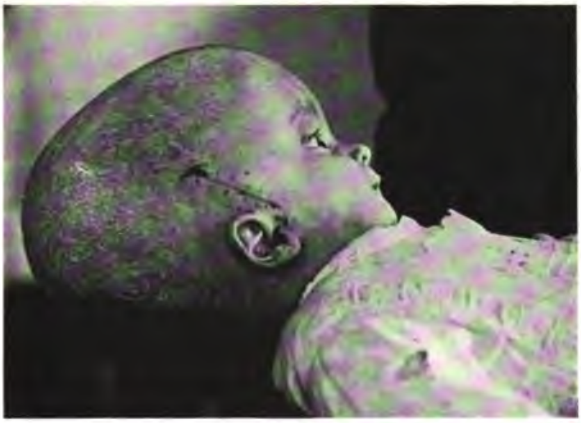


FIG. 196.—No. 966. Ellen, 8 months of age. An advanced condition of external hydrocephalus resulting from an extensive supracortical hemorrhage at the time of instrumental birth; several convulsive seizures occurred within the first three days following birth but not associated with an increased temperature; a lumbar puncture at this time removed bloody cerebrospinal fluid but not under high pressure (undoubtedly due to the compensatory enlargement of the head which was of normal size at birth). The head gradually enlarged after one month of age, most probably due to an increasing blockage of the excretion of the cerebrospinal fluid into the supracortical veins and sinuses as the result of the connective tissue formation and organization of the layer of supracortical hemorrhage. A right subtemporal decompression with drainage by means of several linen strands at seven months of age has obtained a marked improvement; the ventricles were not dilated.

176.—Chronic severe brain injury at birth, associated with a depressed fracture of the vault, a right spastic hemiplegia and with convulsive seizures; a definite increase of the intracranial pressure. A modified left subtemporal decompression. Excellent recovery.

No. 64.—Francis. Twenty-six months. White. U. S.

Admitted April 15, 1914—26 months after birth and injury. Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Operation April 16, 1914. Left subtemporal decompression and removal of depressed area of bone.

Discharged April 26, 1914—10 days after operation.

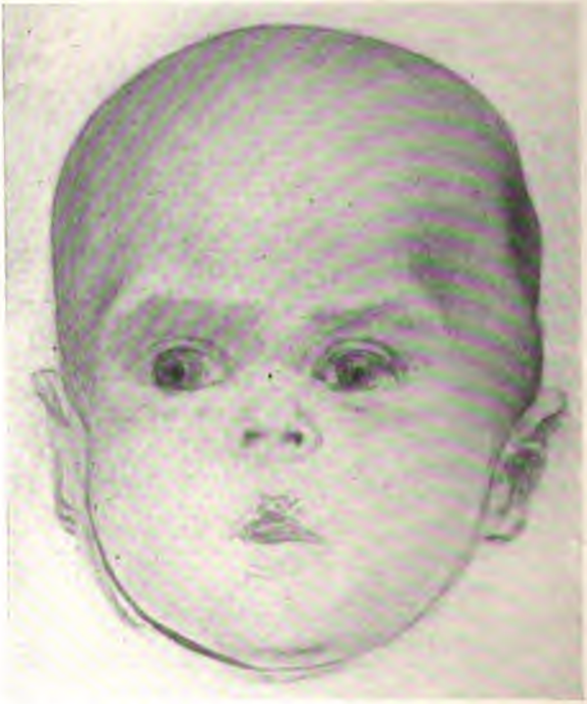


FIG. 197.—Depressed fracture of the left parieto-squamous area following an instrumental delivery is a child two years of age; an increased intracranial pressure associated with a right spastic hemiplegia and convulsive seizures. Excellent recovery following a left subtemporal decompression.

Family history negative; four older children all living and well.

Personal History.—Fifth child, nine months pregnancy, difficult labor requiring instruments; weight 9 lbs. Immediately after delivery, a large depression of the left lower parietal area was observed—the diameter being 2 inches and its depth about ½ inch. No attempt was made to elevate the bony depression, as it was thought that it would disappear itself—like many "ping-pong" depressions of the vault of newborn children. Eighteen hours after birth, a convulsive twitching of right side of face and of right arm occurred and continued every half hour for 2 or 3 minutes; 28 hours after birth, a general convulsive seizure with loss of consciousness followed one of these localized convulsive twitchings of the right face and of the right arm;

during the following week, five general convulsive seizures occurred and then gradually the convulsive twitchings of the right side of face and the right arm subsided and did not reappear until a general convulsive seizure occurred during the tenth month after birth and the last one during the eighteenth month after birth—that is, 8 months ago. During this entire period, the depressed area of the left parieto-squamous bone remained the same. At six months after birth, it was observed that there was a definite weakness of the right arm and of the right face; the child did not hold its head up until 11 months after birth, did not sit up until 16 months after birth and it only began to walk 4 months ago—that is, at 22 months of age. During this period there was a definite spastic paralysis of the entire right side of the body—particularly of the right arm and of the right leg, which were held in the typically flexed and stiff postures. The child was considered normal mentally in its speech and behavior. The treatment of the child had consisted of daily massage during the past six months.

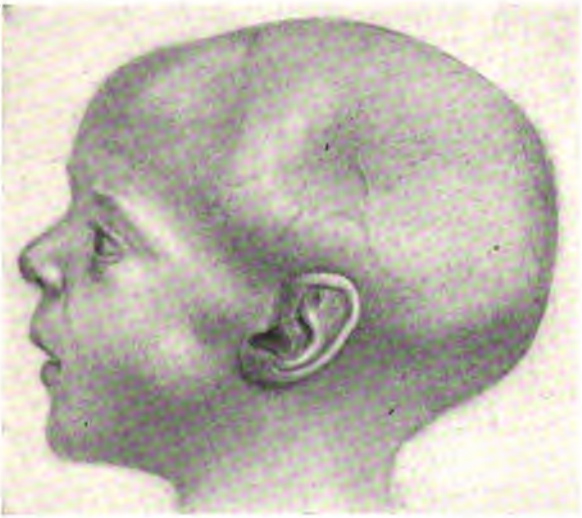


FIG. 198.—Side view of the same patient, having a depressed fracture of the left vault and a supracortical hemorrhage at the time of birth. A left subtemporal decompression afforded almost immediate improvement.

Examination upon admission (26 months after cranial injury).—Temperature, 98.6°; pulse, 88; respiration, 26; blood-pressure, 106. A well-nourished child. A definite retardation mentally is elicited by the special tests; react rather sluggishly. In the left parieto-squamous area overlying the left parietal crest is a definite depressed area of almost 3 inches in width and at least one-half inch in depth; no tenderness or pulsation upon palpation (Figs. 197 and 198). Typical right spastic hemiplegia involving the right arm, the right leg and the cortical type of paralysis of the right side of the face; no sensory impairment, however, could be elicited. When walking, child drags right leg and walks upon the toes. A definite spinal curvature—a right dorso-lumbar compensatory scoliosis. Hearing apparently negative. Pupils equal and react to light normally. No strabismus or nystagmus ascertained. Reflexes: patellar exaggerated, right more than left; right exhaustible ankle clonus and right Babinski; abdominal reflexes—right less active than left. Fundi: retinal veins dilated; nasal margins of both optic disks blurred by edema; the optic disks themselves are possibly paler than normal. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 16 mm.); Wassermann test was reported negative later. X-ray (Doctor A. J. Quimby)—"a depression of the vault of the left parieto-squamous area—2½ inches in diameter and almost 2 cm. in depth; no linear fracture observed."

Treatment.—As the increased intracranial pressure appeared higher than due merely to the depressed bone, a removal of the depressed area of bone was advised by means of a high left subtemporal decompression.

Operation (April 16, 1914—26 months after injury).—A left subtemporal decompression and removal of the depressed area of the vault: the usual vertical incision extended one inch higher to the upper edge of the bony depression; Doyen perforator and burr used to make a small opening at the edge of the bony depression, and the depressed bone removed and also the usual area of the squamous bone rongeured away; the underlying dura was thickened, especially beneath the depressed bone, and it bulged outward under high tension upon removing the bone. Upon opening the dura, which was very much thickened, the cerebrospinal fluid spurted to the height of 2 inches and, upon enlarging the dural opening, the underlying "wet" edematous cortex tended to protrude but did not rupture, owing to the rapid escape of much cerebrospinal fluid. No subdural hemorrhagic cyst observed, but along the cortical vessels in the sulci was a hazy whitish thickening—undoubtedly due to a former supracortical hemorrhage. The cerebral cortex beneath the depressed area of the vault was in no way more damaged than the adjacent areas as described above. No signs of cortical laceration or cortical hemorrhage observed. Normal pulsation at end of operation. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes—Uneventful convalescence in that operative incision healed *per primam;* it was observed on the fourth day after operation that the right arm and right leg were definitely less stiff and less spastic than before the operation and the child could move them much more freely and less awkwardly; this improvement rapidly continued.

Examination at discharge (10 days after operation).—Temperature, 98.6°; pulse, 84; respiration, 24; blood-pressure, 108. No complaints except for soreness at site of operation. Decompression area bulges slightly beyond the flush of scalp; normal pulsation. Child walks with a much less limp than before the operation, the heel touches the floor and the leg is n
dragged; the flexor contraction of the right arm is not so marked; h
to use the hand much more freely than formerly. Pupils equal and
light normally. Reflexes: patellar, right more active than left; r
haustible ankle clonus and right Babinski still persist; abdominal re
right less active than left. Fundi: retinal veins enlarged; lower na
gins of both optic disks slightly blurred by edema.

Treatment.—The usual hygienic measures advised, including a thorough daily massage of the right side of body.

Examination (May 6, 1914—21 days after operation).—No complaints. Operative area is flush with the surrounding scalp; pulsates normally. Child can now walk with no limp at all, although rather awkwardly; right arm and right hand are now held down at the side in a much more normal position; child is using the hand much more naturally. Reflexes: patellar, right more active than left; no ankle clonus and no Babinski can be elicited; abdominal reflexes, right still slightly depressed. Fundi: retinal veins slightly enlarged; nasal margins are now clear and distinct.

Examination (June 9, 1914—54 days after operation).—No complaints. Decompression area possibly slightly depressed beneath flush of scalp; normal pulsation. Complete recovery of the use of right arm and right leg; no noticeable stiffness and spasticity of the right arm and right leg; child walks with no limp and can run in a normal manner; he is now using the right hand almost as freely as the left, although the child was formerly considered as being left-handed. Parents have noticed a definite mental and emotional change in that the child is brighter and is less irritable. Reflexes: patellar, right more active than left; no ankle clonus nor Babinski; abdominal reflexes—right slightly depressed. Fundi: retinal veins possibly larger than normal; all details of both optic disks clear and distinct; both optic disks slightly paler than normal.

During the last 5 years, this child has been repeatedly examined and the result has been a most gratifying and satisfactory one; no convulsive seizures have occurred, and the physical, mental and emotional condition of the child may be considered that of a normal child. (Fig. 199 pictures child at five years of age and 3 years after operation.) At the last examination, on April 6, 1919—60 months after operation, the operative area was depressed and very much narrowed, due to new bone formation about the periphery so that it was difficult to perceive any pulsation. The reflexes were active but equal and otherwise negative, and the fundi were negative except for a slight pallor of the temporal margins of both optic disks. The mental and emotional reactions were normal.

Remarks.—The condition of this child was one of typical spastic hemiplegia so frequently observed following an intracranial hemorrhage at the time of birth and without the complication of a depressed fracture of the overlying vault; it was, however, this bony depression which made the diagnosis such a simple one clinically and there could be no doubt that the depressed area of bone overlying the left cerebral motor cortex was the cause for the motor impairment of the right side of the body; in the vast majority of the patients, however, having a spastic paralysis and due to a supracortical hemorrhage at the time of birth, there is no overlying depressed fracture of the vault, but that does not mean that there cannot be a definite cerebral compression due to a supracortical hemorrhage and without the complication of a depressed fracture of the vault; these are the patients having no depressed fracture of the vault which have been so overlooked in the past, and yet the diagnostic signs of the condition are now so easily obtained by means of the ophthalmoscopic examination of the fundi

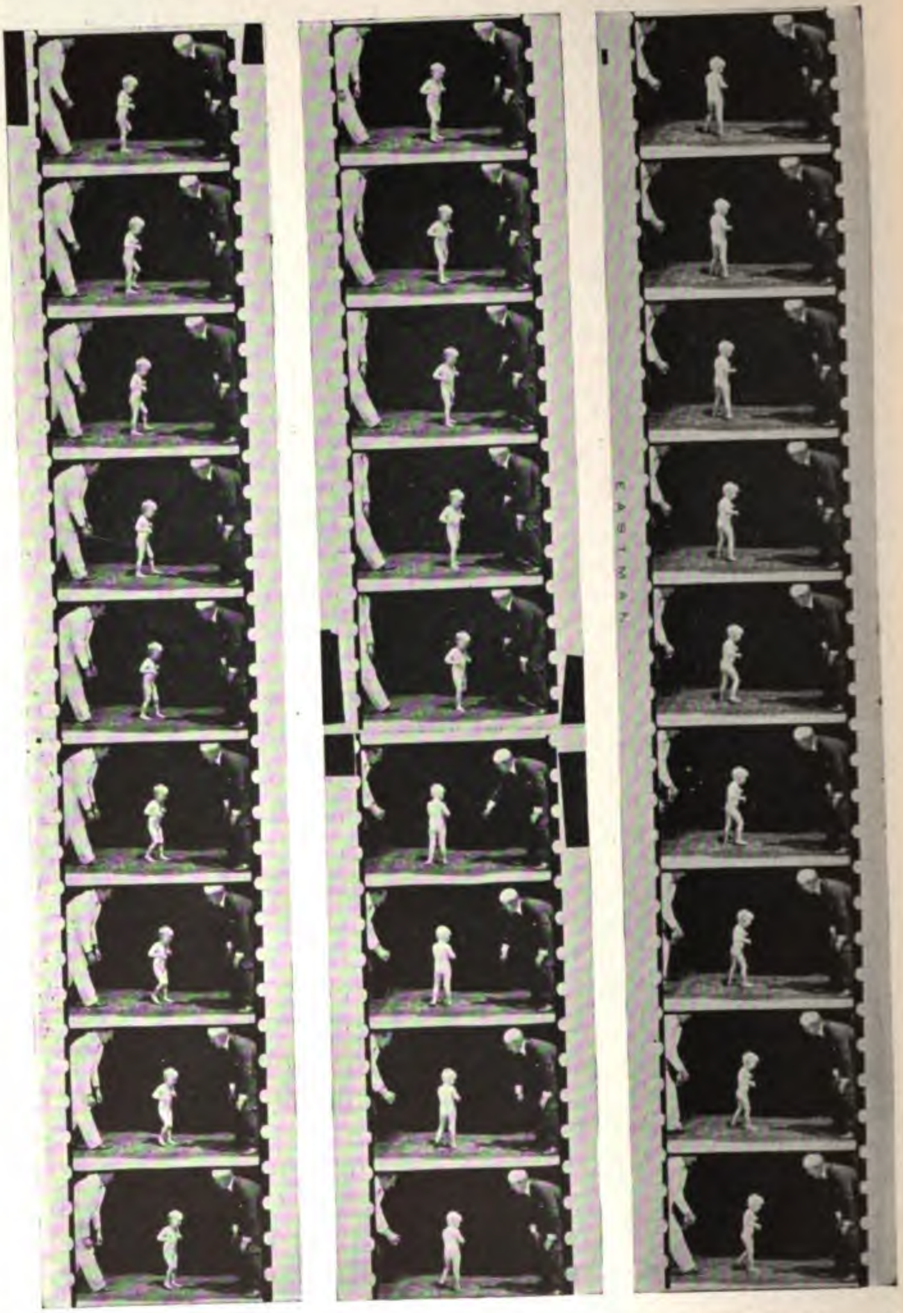


FIG. 199.—The patient at five years of age and three years after the operation of left subtemporal decompression and drainage for the condition of a supracortical hemorrhage at birth associated with a depressed fracture of the left parieto-squamous area; the resulting right spastic hemiplegia has disappeared while the convulsive seizures have not returned since the operation.

and the registration of the pressure of the cerebrospinal fluid by means of the spinal mercurial manometer and thus the patients having an increased intracranial pressure can easily be differentiated from those other patients having no increased intracranial pressure and naturally not to be benefited by any operative procedure upon the skull. The depressed fracture of the vault of this patient merely facilitated the diagnosis from a superficial standpoint, and yet in this patient there were the definite signs of an increased intracranial pressure and of such a height that it was considered advisable to open the dura and to let it remain open rather than the mere elevation or removal of the bony depression.

The operative findings of organization of the former supracortical hemorrhage in the sulci about the cortical vessels confirmed the opinion of a former subdural hemorrhage, and it is this partial blockage of the stomata of exit of the cerebrospinal fluid into the cortical veins that produces the "wet" edematous condition of the cortex and permits the persistence of an increased intracranial pressure in the form of a mild external hydrocephalus, which is the secondary pathology in many of these patients.

The almost immediate and excellent improvement obtained in this patient is not an unusual one; the ultimate result—ten, fifteen and twenty years from now, however—may not be that of an entirely normal individual—especially in his emotional reactions; and then again, the great danger of epileptiform seizures occurring later in life and due to the irritative presence of the residue of the former supracortical hemorrhage in the sulci and about the cortical vessels; the operative drainage of this mild condition of external hydrocephalus undoubtedly lessens the irritative effect of the condition, and it is hoped entirely so; a longer period of time, however, must elapse before it is possible to state a final opinion.

As in the other patients having an intracranial hemorrhage at the time of birth and usually following a difficult labor with and without the use of instruments, the ideal time for the drainage of the supracortical blood and the relief of the increased intracranial pressure is as soon as possible after the birth—upon the first day, second day, third day and as early as possible, when the blood itself can be drained in fluid form, and just as in this patient an earlier operation would have afforded not only a greater chance of immediate relief and of ultimate complete recovery, but it would have avoided the temporary paralysis and the mental and emotional retardation which had occurred during the time preceding the operation. The operative indication in this particular patient was so self-evident from even a manual examination of the skull that it may be considered one of the neglected patients.

CASE 177.—Chronic severe brain injury at birth associated with a supracortical hemorrhage, a right hemiplegia and an occasional Jacksonian convulsion of the right arm and right leg; an increased intracranial pressure. Left subtemporal decompression and drainage. Marked improvement.

No. 329.—Frances. Two years. White. U. S.

Admitted September 15, 1915—24 months after birth and injury. Polyclinic Hospital. Referred by Doctor M. Allen Starr.

Operation September 22, 1915—7 days after admission. Left subtemporal decompression and drainage.

Discharged October 2, 1915—10 days after operation.

Family history negative. Parents and their relatives all right-handed.

Personal History.—First child, full term, apparently normal labor and no instruments required; weight, seven and a half pounds at birth; rather difficult to resuscitate, but not more so than in many babies; no convulsive seizures or twitchings. The child was considered a normal one until 8 months of age, when it was observed for the first time that the right arm and right leg were slightly more stiff than the left arm and left leg; also the child was not holding its head up as early as it should; it was at this time, that momentary twitchings of the right arm were first observed—not more than two or three a week and lasting for only several seconds; no loss of consciousness, although mother states that sometimes child "seemed to stare," while the twitching of the fingers of right hand continued. The general condition, however, gradually improved so that the child was able to sit up at fourteen months of age and began walking at nineteen months of age (5 months ago). Speech has been retarded, so that at present the child is only able to say "mamma" and "no." Patient has had daily massage and exercises for several months. During the past 6 months, a slight convulsive twitching of the right arm and frequently of the right leg occurred for a period of 5 to 6 seconds, and it has been observed that while the twitching continued the child would have a very "vacant" expression, but no complete loss of consciousness. Persistent constipation requiring medicines and enemata. No history of injury to the head; always well and strong.

Examination upon admission (24 months after birth and injury).—Temperature, 98.6°; pulse, 80; respiration, 26. Well-developed and nourished. Definite spastic paralysis of the right arm and of the right leg, also right facial weakness of the cortical type; no impairment of sensation can be elicited. Child holds right arm in the slightly flexed and pronated manner typical of spastic paralysis; right leg is slightly flexed at the knee and the child walks chiefly upon the toes of the right foot—a mild talipes equinus. Mental condition rather retarded—the Simon-Binet test registering her age as less than two years. Is able to say "mamma," "no" "Fran" and "I," but with indistinctness. No marked impairment of swallowing. Pupils equal and react to light normally. Reflexes: patellar exaggerated. right more than left; no ankle clonus but right Babinski: abdominal reflexes—right depressed; reflexes of right arm increased. Fundi—retinal veins slightly enlarged; nasal margins of both optic disks blurred by edema. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 14 mm.); Wassermann test negative and cell count was 5 cells per c.mm. X-ray (Doctor A. J. Quimby)—"no abnormalities observed."

Treatment.—The presence of an increased intracranial pressure associated with a right spastic hemiplegia and with the history of convulsive twitchings of the right arm and at times of the right leg, indicated the advisability of a left subtemporal decompression in the belief that a lowering of the general intracranial pressure would permit a definite lessening of the spasticity and an improvement of the mentality and also afford the patient a chance of avoiding the great danger of future epilepsy.

Operation (24 months after birth and injury).—Left subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications; the bone was unusually thin, almost like tissue paper, and most probably due to the prolonged increase of the intracranial pressure. Dura whitish, thickened and under tension; upon incising it, clear cerebrospinal fluid spurted to a height of 2 cm. and upon enlarging the dural opening a very edematous tense cortex protruded but did not rupture. Along the vessels in the sulci was a whitish, cloudy, connective-tissue formation—the organization of a former subarachnoid and supracortical layer of hemorrhage. The convolutions themselves appeared normal, although possibly paler than usual. At the end of the operation, the cortex became more relaxed and pulsated almost normally following the loss of much cerebrospinal fluid. Usual closure with 2 drains of rubber tissue inserted. Duration, 35 minutes.

Post-operative Notes.—Uneventful recovery; operative incision healed *per primam*. Decompression area bulged beyond the flush of scalp at the time of the patient's discharge—10 days after operation.

Treatment.—The usual massage and exercises advised just as before the operation.

Examination (May 20, 1916—8 months after operation).—Child has made a marked improvement since the operation in that the stiffness of the right arm and of the right leg has markedly lessened, the child now using the arm more freely and holding it more naturally, and she is able now to walk more upon the right heel rather than upon the toes of the right foot. The mentality has very much improved in that she is more alert and is more interested in things: she is now able to say a number of sentences and with distinctness; she has not had a convulsive twitching since the operation. Decompression area slightly depressed beneath the flush of scalp; pulsates normally; bony opening somewhat narrower, due to new bone formation about the periphery. Patient walks with only a slight limp upon the right foot—hardly noticeable. Reflexes—patellar active, right more than left; no ankle clonus but right Babinski still persists; abdominal reflexes—right less active than left; reflexes of right arm more active than of the left arm. Fundi—retinal veins of normal size; details of both optic disks clear although a small amount of new tissue formation is present at the lower nasal margins of both optic disks. Massage and exercises continued as before.

Last Examination (April 16, 1919—43 months after operation).—The condition of the child has continued to improve, and if it were not for the very slight awkwardness of the right arm and of the right leg, she might be considered a normal child in every way; mentality excellent and no retardation or impairment elicited. Decompression area is depressed below the flush of scalp; normal pulsation palpable. Reflexes: patellar—right greater than left; right Babinski persists; abdominal reflexes—right possibly less than left; reflexes of right arm more active than those of left. Fundi—negative, except for new tissue formation along the lower portions of the nasal margins of both optic disks. Child walks, runs and plays with little or no impairment of the right side of the body with the exception of a slight awkwardness in the use of the right hand, and its impairment is only elicited by special tests.

Treatment.—The daily massage and exercises are to be continued as before.

Remarks.—It is the marked improvement occurring in patients as in this child which impresses one with the advisability of performing the operation in these selected children having an increased intracranial pressure due to a hemorrhage at the time of birth—and as soon as possible after the birth and the injury; if such a marked improvement can be obtained two years after the injury, surely a greater improvement and even the avoidance of the results of the intracranial hemorrhage and its increased intracranial pressure in the later spasticity and mental retardation would have been obtained, if the operation had been performed within several days after the birth of the child when the drainage of the blood itself would then have been possible. Later, and surely after one year of age, all that the operation can accomplish is to lower the increased intracranial pressure both by offsetting the pressure effects of the hemorrhage and its resulting cystic and fibrous tissue formation and, more important, to afford drainage to the partially blocked cerebrospinal fluid as a result of the hemorrhage in the sulci forming connective tissue organization about the cortical veins and in this manner the condition of mild external hydrocephalus is developed. Naturally, the ideal treatment of these selected patients is the drainage of the blood itself within a few days after the birth of the child and the improvement to be obtained in these older patients merely emphasizes the necessity of more careful examinations and more accurate diagnosis at the time of the birth of the patient—rather than months and even years later, when the spasticity and mental impairment are well developed and when the probability of obtaining a normal child is practically *nil* and all that can be expected even with the most successful of cases is merely an improvement and an approximation to normality. In all newborn babies, whether the labor is apparently a normal one or whether it is a difficult one with and without the use of instruments, if the child does not appear to behave as it normally should by being rather drowsy or stuporous and surely in the presence of convulsive twitchings, or if it should be a "blue" baby, then careful ophthalmoscopic examinations should be made and repeated lumbar punctures, if necessary, to determine the pressure of the cerebrospinal fluid and the presence or not of blood in it; it is only by this means that a large number of babies having an intracranial hemorrhage at the time of birth will be diagnosed early and the appropriate treatment of spinal drainage or the cranial decompression and drainage instituted

Although the improvement in this patient has been excellent a
cessation of the convulsive twitchings most hopeful, yet it will be ne
for a period of years to elapse before it can be definitely stated t
child is beyond the risk of epilepsy; it will also be necessary to w
this child enters into active life with its strain and stress before it
definitely ascertained, whether its reactions are as normal as they sho

in fact, it would be better judgment that this child should be restricted in its life and not be permitted to lead a too strenuous existence, filled with excitement and unnecessary cares and worries.

Case 178.—Chronic severe brain injury at birth associated with a supracortical hemorrhage and a resulting spastic diplegia; an increased intracranial pressure. Right subtemporal decompression. Marked improvement.

No. 1005.—Ruth. Two and a half years. White. U. S.

Admitted July 14, 1917—30 months after birth and injury. Polyclinic Hospital. Referred by Doctor B. H. Whitbeck.

Operation July 19, 1917. Right subtemporal decompression and drainage.

Discharged July 30, 1917—11 days after operation.

Family history negative; two older children living and well.

Personal History.—Third child, full term, face presentation, difficult labor requiring instruments; rather bluish at birth, requiring prolonged efforts to resuscitate her; no convulsive twitchings. Apparently a normal child and was so considered until 12 months of age, when it was realized that the child was not holding her head up as well as normally and could not sit up alone. At sixteen months of age, it was observed for the first time that both legs were rather stiff and adducted and that the thighs could not be adducted unless with effort; also that the left foot "turned in." Vigorous daily massage and passive exercises were administered and at the eighteenth month the child succeeded in sitting up alone. The physical development was slow and although the child was well-nourished and apparently well-developed, yet she has never been able to walk and does not attempt to crawl about like other children. Mentality only slightly impaired. No convulsive seizures. Patient has received daily massage during the past 18 months, but with little or no improvement.

Examination upon admission (30 months after birth and injury).—Temperature, 98.6°; pulse, 82; respiration, 26. Well-nourished child. Unable to walk due to the stiffness of both legs which are held slightly flexed and adducted—"scissors" type; when held up, patient stands upon the toes. Arms slightly stiff but much less so than the legs. Mentality only slightly impaired—Simon-Binet tests registering her age as of 2 years. No history of convulsive seizures or twitchings. Patient can sit up alone but with some difficulty. Pupils equal and react normally. No nystagmus. Reflexes: all exaggerated; patellar very active, left more than right; no ankle clonus but double Babinski; abdominal reflexes present but depressed equally; both arm reflexes slightly increased. Fundi—retinal veins slightly enlarged with thickened walls; nasal margins of both optic disks blurred by edema and physiological cups are both shallow from new tissue formation; both optic disks are possibly paler than normally. Lumbar puncture—clear cerebrospinal fluid under increased pressure (16 mm.); Wassermann test negative and cell count was 3 cells per c.mm. X-ray (Doctor W. H. Stewart) —"negative."

Treatment.—The spastic diplegia and slight mental retardation associated with an increased intracranial pressure made it advisable to perform a subtemporal decompression in the belief that a lowering of this increased intracranial pressure would permit a definite lessening of the spasticity and afford the patient a chance of improvement both physically and mentally; the absence of convulsive seizures made the case a more hopeful one from the standpont of the ultimate prognosis; the left side of the body being more impaired, a right subtemporal decompression was performed.

Operation (30 months after birth and injury).—Right subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications. Dura whitish, thickened and tense; upon incising it, clear cerebrospinal fluid welled out in large quantity, forming a "puddle"; the underlying edematous cortex protruded but did not rupture, as the amount of the escaping cerebrospinal fluid was profuse. No gross signs of former supracortical hemorrhage observed, except along the cortical veins in the sulci there was a whitish induration and a sort of cystic formation lying upon the cortex and beneath the arachnoid; upon puncturing the latter, much straw-colored fluid escaped, permitting the cyst to collapse and the underlying cortex to rise. At the end of the operation, the cortex pulsated almost normally, but the intradural pressure was still increased. Usual closure with 2 drains of rubber tissue inserted. Duration, 30 minutes.

Post-operative Notes.—Uneventful recovery; wound healed *per primam*, and the area bulged tensely but pulsated. Before the child left the hospital at the end of ten days, a definite lessening of the spasticity of both legs was observed—and possibly of the left more than of the right leg.

Treatment.—The parents were advised to continue the daily massage and exercises as before the operation.

Examination (May 10, 1918—10 months after operation).—A very marked improvement of the condition has occurred since the operation; the spasticity of both legs has so lessened that they no longer are adducted and the child is able to crawl and is trying to stand alone; she no longer stands upon the toes. Use of both arms has also improved so that they are now practically normal. Much brighter mentally and the child is able to talk as a normal child. Decompression area remains flush with the surrounding scalp; normal pulsation. Reflexes—patellar active but equal; no ankle clonus but double Babinski persists; abdominal reflexes present and equal; reflexes of both arms active but equal. Fundi—retinal veins slightly enlarged; slight blurring of the lower nasal margins of both optic disks.

Treatment.—The daily massage and exercises to be continued; it is hoped that the decompression area will become depressed beneath the flush of the scalp and thus indicate the permanent lessening of the intracranial pressure; if this does not happen within a year, it will probably be necessary to perform a left subtemporal decompression and drainage in order to permit the greatest ultimate improvement to occur. (The parents, however, are so pleased with the continued improvement of the child that a second
is not desired.)

Last Examination (June 10, 1919—23 months after operati
marked improvement continues so that now the child is able to
braces devised by Doctor Whitbeck and in every way it would see
child is approximating normality, and especially is this true o
tality. The decompression area is becoming slightly depressed a

believed that a bilateral decompression will not be necessary. The use of the arms is excellent. Reflexes: increased but equal; double Babinski still persists; arm reflexes negative. Fundi—retinal veins possibly slightly enlarged; an indistinct blurring of the lower nasal margins of both optic disks persists; the new tissue formation remains as before the operation. The photograph shows the present improved condition of this patient (Fig. 200).

Remarks.—The operative findings and the rapid subsequent improvement of the condition of this patient are very impressive; if, at the time of birth and when it was observed that the child was cyanotic, requiring vigorous resuscitation, a lumbar puncture had been performed disclosing an increased pressure of the cerebrospinal fluid and the presence of blood—if then there had been performed a decompression and drainage of the free supracortical blood, not only would this physical and mental impairment have been avoided but the child could have become a normal child. If only an increased pressure was ascertained at the time of birth and little or no blood found in the cerebrospinal fluid at lumbar puncture, then repeated lumbar punctures might have in themselves been sufficient to lessen the intracranial pressure by the drainage of the excess cerebrospinal fluid and any free blood, and in this way a normal child be obtained without the added risk of a cranial operation and drainage.

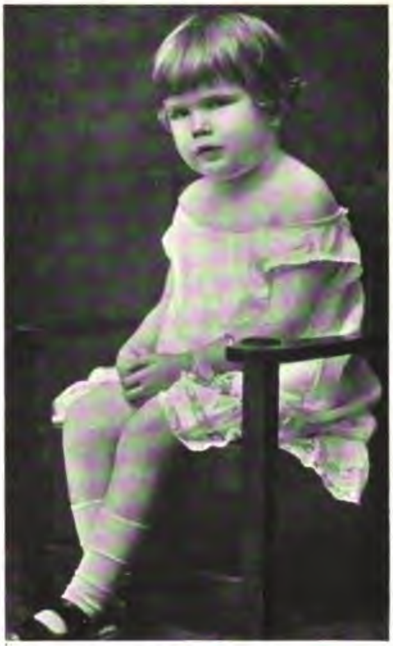


FIG. 200.—The present improved condition of the patient—an almost normal mentality, and a spasticity of only slight degree.

It will be necessary to wait in this patient for a period of years to elapse in order to estimate the ultimate improvement to be obtained, and this is true of all these children upon whom the cranial operation of decompression and drainage has been performed—the first operation being in June, 1913; so far, the younger the child when operated upon, the greater the improvement.

CASE 179.—Chronic severe brain injury at birth associated with a supracortical hemorrhage and a resulting spastic diplegia, mental retardation and convulsive seizures; an increased intracranial pressure. Left subtemporal decompression. Definite improvement.

No. 91.—Elsie. Six years. White. U. S.

Admitted November 15, 1914—6 years after birth and injury. Polyclinic Hospital. Referred by Doctor J. A. Bodine.

Operation November 24, 1914. Left subtemporal decompression and drainage.

Discharged December 8, 1914—14 days after operation.

Family history alcoholic; sister of father was epileptic; otherwise negative; three older children living and well; no miscarriages for mother.

Personal History.—Fourth child, full term, normal labor; apparently normal after birth until the third day, when a series of general convulsions occurred—ten on the third, six on the fourth day and three on the fifth day after birth; these general convulsive seizures gradually lessened in frequency until the third year when a convulsion occurred on the average of one each week; after the third year and until admission to the hospital, a general convulsive seizure occurred on an average of one each month; no localizing signs at any time. General retardation of the physical development of the child in that it could not sit up until sixteen months of age, only learned to creep at 2 years of age and was unable to walk until 3 years of age. The definite stiffness of both legs and more of the right leg was not noticed until the child was 12 months of age; this spasticity gradually increased in spite of daily massage, so that when the child finally learned to walk at 3 years of age it did so with a typically spastic gait—the right side of the body being much worse than the left. The child has always carried the right arm flexed at the elbow and at the wrist with the hand held in the position of pronation. The child finally learned to talk in monosyllables at 3 years of age and the speech has gradually improved. Definite retardation of mentality, so that it has been impossible to send the child to school. Very irritable, having a "bad temper," and she is very difficult to manage—especially after the convulsive seizures which occur now on an average of one each month. During the past two years, the patient has received massage daily and triple bromides.

Examination upon admission (6 years after birth and injury).—Temperature, 98.6°; pulse, 84; respiration, 26. Rather poorly developed and nourished. A condition of mild spastic diplegia with the right side more spastic than the left; both thighs adducted and flexed at the knees, right more than left; child walks upon the toes of the right foot in the position of talipes equinus; the right arm is held flexed at the elbow with the wrist and the hand pronated—the typical position assumed by patients having the condition of spastic paralysis (Fig. 201). The left arm and left leg are only slightly spastic and with only mild muscular contractions. Mentality is that of a child of four years; irritable and rather surly. (Last convulsive seizure occurred 10 days ago; it began with a typical epileptic cry to be followed by a characteristic general convulsive seizure with no localizing signs; the tonic and clonic muscular contractions lasted for almost 3 minutes.) Right facial weakness of the cortical type (the upper third of right side of face not being involved). Pupils equal and react to light normally. Reflexes: patellar—both exaggerated, right much more than left; right ankle clonus and double Babinski; reflexes of both arm
right more than left. Fundi—retinal veins dilated; nasal halv
optic disks obscured by edema; both physiological cups shallow fro
tous tissue. Lumbar puncture—clear cerebrospinal fluid under
pressure (approximately 18 mm.); Wassermann test negative an
was 4 cells per c.mm. X-ray (Doctor A. J. Quimby)—"no a
of the skull is observed, except a thinning of the vault poste

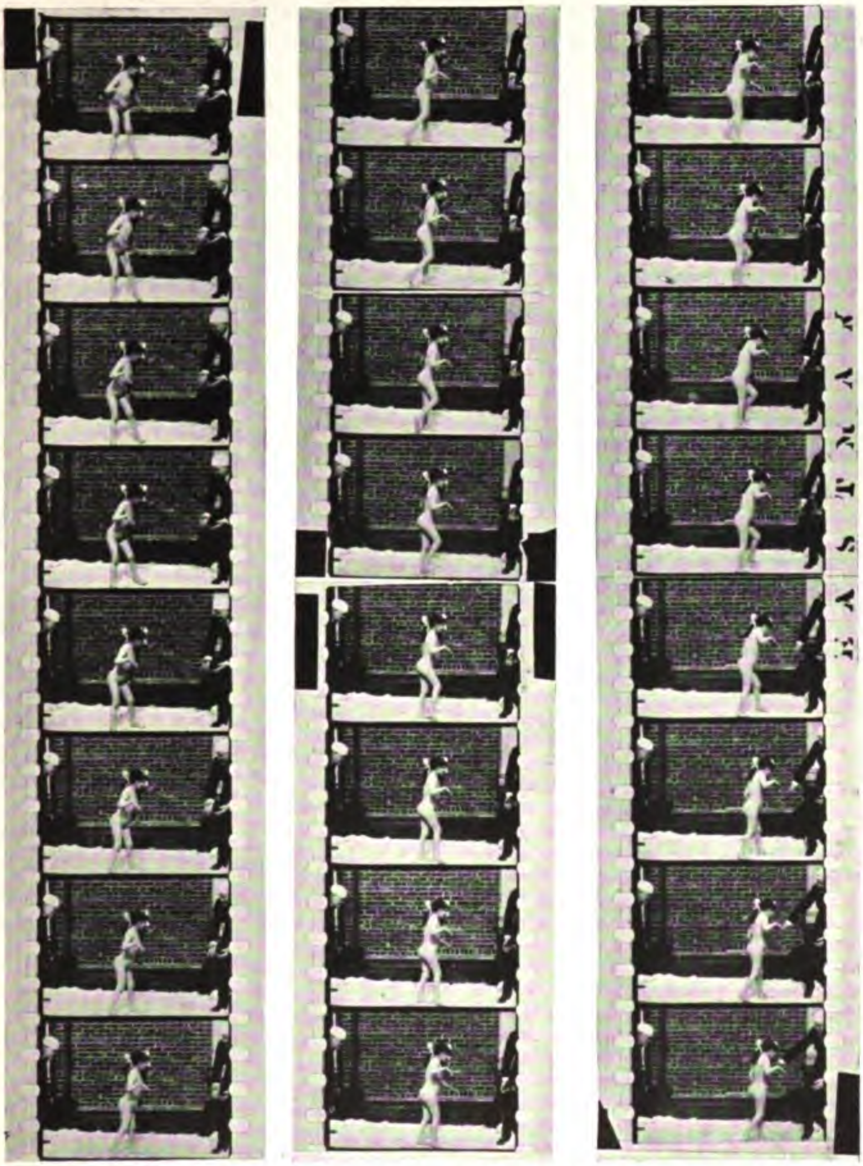


FIG. 201.—The patient, six years of age, having a right spastic hemiplegia (affecting chiefly the right arm), following a supracortical hemorrhage at the time of birth with resulting mental retardation and convulsive seizures. Marked improvement following a left subtemporal decompression and drainage at six years of age.

anterior fontanelle and very probably due to a prolonged increase of the intracranial pressure." (Fig. 202.)

Treatment.—The occurrence of convulsive seizures on the third day after an apparently normal birth and the later development of the condition of spastic diplegia with mental retardation in the presence of a marked increase of the intracranial pressure—this clinical picture made most probable the diagnosis of an intracranial hemorrhage at the time of birth and the resulting increased intracranial pressure as being due to this former hemorrhage with a subsequent partial blockage of the excretion of the cerebrospinal fluid—a mild condition of external hydrocephalus. A left subtemporal decompression and drainage was advised as a means of lowering this increased intracranial pressure and thus affording this patient a definite chance of improvement.

Operation (6 years after birth and injury).—Left subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications. Dura thickened, whitish and tense; upon incising it, clear cerebrospinal fluid spurted to a height of 2 cm., exposing a very edematous cortex. Above the Sylvian fissure was an extensive cystic formation compressing the underlying cerebral convolutions and extending upward beyond the upper edge of the dural opening; numerous adhesions between the outer wall of this cyst and the overlying dura were present. About the vessels in the sulci, and especially above the Sylvian fissure, was a cloudy induration—the result of a former subarachnoid hemorrhage. Much cerebrospinal fluid escaped during the operation so that the cerebral pulsation became normal. Usual closure with 2 drains of rubber tissue inserted. Duration, 45 minutes.

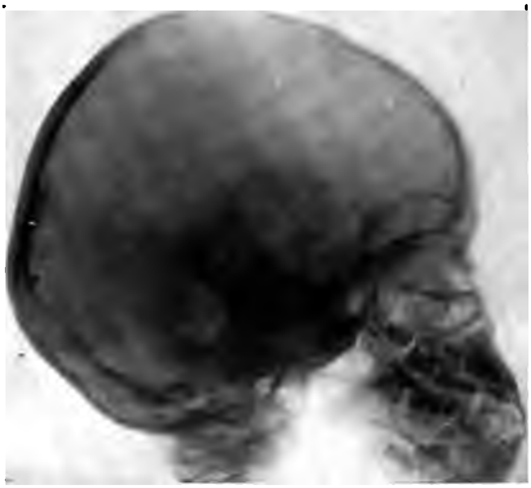


FIG. 202.—Pressure atrophy of the inner table of the vault just posterior to the anterior fontanelle and most probably due to a prolonged increase of the intracranial pressure.

Post-operative Notes.—Uneventful operative recovery; incision healed *per primam* and at discharge on the fourteenth day after operation, the decompression area bulged only slightly beyond the flush of scalp; no convulsions occurred during the hospital residence.

Examination (May 6, 1916—18 months after operation).—A definite improvement has occurred both mentally and physically in that the left side of the body is practically normal, whereas the spasticity and contractures of the right arm and of the right leg have so lessened that these extremities can be used awkwardly; the mentality has definitely improved but comparatively less than the physical condition. No convulsive seizures, however, have occurred since the operation. Less irritable, and mother states that the child is now "a pleasure to live with." Reflexes: patellar—right exaggerated; exhaustible right ankle clonus and double Babinski still persist; abdominal reflexes—right possibly less active than left; deep reflexes of right arm increased. Fundi—retinal veins slightly enlarged; no edematous blurring of the details of either optic disk; connective-tissue formation is present as before the operation. Vision apparently 16/20 in each eye. Decompression area depressed beneath the flush of the scalp and pulsates normally.

Last Examination (April 22, 1919—53 months after operation).—This patient has continued to improve so that now there is only a slight limp of the right leg and a definite awkwardness and spasticity in the use of the right arm; the mentality has steadily improved, although the mental age is registered as being 2 years below normal; no convulsive seizures have occurred. Reflexes: patellar—right much greater than left; no ankle clonus but double Babinski; deep reflexes of right arm are more active than of left. Fundi—retinal veins of normal size; all details of both optic disks clear and distinct except for a small amount of new tissue formation at the margins and in the physiological cups (as at examination before the operation). Decompression area remains depressed beneath the flush of scalp but pulsates normally; operative opening smaller from new bone formation about the periphery.

Remarks.—The cessation of the convulsive seizures to the extent that not one convulsion has occurred since the operation is most impressive regarding the apparent beneficial effect of the lowering of the increased intracranial pressure—even in the presence of numerous adhesions between the dura and the underlying cerebral cortex. Naturally, these adhsions at the operative site were severed as far as possible and yet beyond the bony edge of the decompression opening, other similar adhesions could be seen extending upward, forward and backward and most probably they were present very extensively over the cerebral cortex, so that it cannot be advocated that the removal of several cortical adhesions was the cause for this marked improvement and cessation of the epileptiform seizures, but rather this improvement is due to the lowering of the increased intracranial pressure alone; in other patients where no cortical adhesions are found, yet the convulsive seizures in these patients may cease entirely following a simple decompression. It is my belief as a result of observing a number of these similar patients that the epileptiform seizures occurring in them are more the result of the original brain injury associated with an increased intracranial pressure, and that cortical adhesions are only a small factor in the condition. A longer period of time must elapse in this patient before it can be stated with any degree of certainty that the convulsive seizures will not recur—in fact, if there is no recurrence before the patient reaches the age of 30 years, then the danger of their reappearance is very slight indeed.

The persistence of the impairment of the right arm and of the right leg is undoubtedly due to a definite damage to the cortical cells underlying the cystic formation over the left cerebral hemisphere, and it will be most probably a permanent one. Clinically, however, this condition of the patient which was at the time of the operation one of spastic diplegia has been changed to one of right spastic hemiplegia of mild degree, and it was the lessening of the general intracranial pressure by means of the left subtemporal decompression that permitted the spasticity of the left arm and left leg to disappear—an impairment due most probably to the mild supracortical lesion over the right cerebral hemisphere; the disappearance of the left Babinski is very interesting and instructive.

Case 180.—Chronic severe brain injury at birth associated with a supracortical hemorrhage, causing a left hemiplegia and convulsive seizures; an increased intracranial pressure. Right subtemporal decompression and drainage. Marked improvement.

No. 334.—Louise. Twelve years. White. School. U. S.

Admitted August 18, 1915—12 years after birth and injury. Polyclinic Hospital. Referred by Doctor T. K. Tuthill.

Operation September 30, 1915. Right subtemporal decompression and drainage.

Discharged October 12, 1915—13 days after operation.

Family history negative; no other children; no miscarriages for mother.

Personal History.—First child, full term, difficult labor, requiring instruments; prolonged efforts to resuscitate the child. It remained drowsy and quiet for three days, when a general convulsive seizure occurred to be followed by daily convulsions of a general character during the following 8 days, when they ceased and did not recur until 3 years ago (when patient was 9 years of age). The temperature had not been increased during the 2 weeks following birth and after the convulsions ceased, the child was considered normal in every way until 14 months of age, when it was observed that the left leg and left arm were not being used as freely as the right leg and right arm, and that there was a definite stiffness of the left leg and left arm; this spasticity of the left side gradually increased until at 2 years of age, there were present the typical flexor contractures and postures of the left side of the body. In other ways the development of the child was delayed in the holding up of the head, the sitting up and the ability of the child to stand and to walk; when the child did finally walk at 3 years of age, she dragged the left foot and walked upon her toes; the left arm was held in the typical position of spastic hemiplegia—flexed at the elbow and at the wrist in pronation. The child was able to talk at 2 years of age, but she did not develop mentally as rapidly as is normal. The usual routine treatment of massage and exercises was administered at the Orthopedic Hospital for a period of years and, although the condition improved, yet there persisted a definite left spastic hemiplegia with mental retardation. Three years ago (when the patient was 9 years of age), a general convulsion began in the left arm and then in the left leg to be followed by complete loss of consciousness and a typical epileptiform convulsive seizure; since that time, these convulsions, usually beginning upon the left side and then becoming general, have continued on an average of 2 or 3 each week, and the patient has become markedly less alert mentally and much more irritable; the left spastic hemiplegia has remained practically the same during the past 6 years. She is referred for treatment of the convulsive seizures.

Examination upon admission (12 years after birth and injury).–perature, 98.6°; pulse, 78; respiration, 24; blood-pressure, 116. nourished girl having a definite left spastic hemiplegia (Fig. 203) retarded mentality; little or no interest in her surroundings and rather childish; she stands upon the toes of the left foot, owing to the contracture of the Achilles tendon (really an anatomical shortening of it due to its prolonged contraction), and the left knee is slightly flexed; the left arm is flexed chiefly at the elbow while the hand is held in a mild position of pronation; left facial weakness of the cortical type (left forehead muscles not involved). Simon-Binet test of mentality indicates her mental age as being 8 years. Slight compensatory dorso-lumbar scoliosis. Hearing negative; otoscopic examination negative. Pupils equal and react normally. Reflexes: patellar—left greatly exaggerated; left patellar and left ankle clonus—the latter being restricted by the Achilles contracture; left Babinski and no right plantar flexion; abdominal reflexes—left absent; deep reflexes of left arm exaggerated. Fundi—retinal veins very much enlarged—right possibly more than left; definite blurring of the nasal margins of both optic disks—possibly more extensive over right disk; newly-formed connective tissue about the margins of both disks and in their physiological cups, so that they are slightly paler than normally—a mild condition of secondary optic atrophy. Vision—16/20 in both eyes. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 20 mm.); Wassermann test negative and cell count was 2 cells per c.mm. X-ray (Doctor A. J. Quimby)—"convolutional markings of the inner table of the vault, especially in the frontal and occipital areas and typical of an increased intracranial pressure" (see Fig. 205).

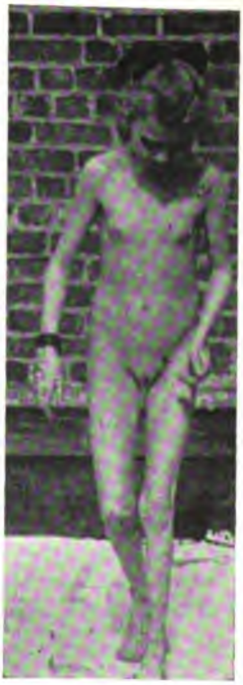


FIG. 203.—Left spastic hemiplegia in a child 12 years of age following a supracortical hemorrhage at the time of difficult birth. Marked improvement following the lowering of the increased intracranial pressure by means of a right subtemporal decompression and drainage even at this late age.

Treatment.—In spite of the advanced age of this patient, yet the presence of a marked increase of the intracranial pressure and the definite localizing signs of a left hemiplegia and the history of the convulsive seizures frequently beginning in the left arm and left leg—these facts made the operation of right subtemporal decompression and drainage advisable in the hope that a definite improvement of the physical and mental condition could be obtained, and most important of all, a lessening of the convulsive seizures. In order to ascertain whether this increased intracranial pressure was the result of the convulsive seizures or whether the increased pressure was possibly a factor in producing the convulsive seizures by increasing the cortical irritability, large doses of triple bromides were administered so that the patient did not have a convulsive seizure during a period of 6 weeks, and yet at the end of this period the increased intracranial pressure registered a height of approximately 20 mm.—that is, just the same pressure as when the convulsive seizures were occurring on an average of two or three each week. It was then decided that this increased intracranial pressure was a very probable factor in producing the convulsive seizures and therefore the operation of right subtemporal decompression and drainage was advised.

Operation (12 years after birth and injury).—Right subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications; much pressure atrophy of the bone, so that it was very thin. Dura thickened, whitish and tense; upon incising it, clear cerebrospinal fluid spurted to a height of 6 inches, revealing a very edematous cortex which protruded and almost ruptured; warm saline cotton compresses held over the opening until sufficient cerebrospinal fluid had drained to permit the cortex to relax and to bulge less tensely. Above the Sylvian fissure was a dark cystic formation lying upon the cortex and having a thickness of one-half inch; it was compressing the underlying cortical cells, and upon puncturing it, a straw-colored fluid escaped, permitting the underlying cortex to rise slightly. The vessels in the sulci, both above and below the Sylvian fissure, were surrounded by a cloudy induration of connective tissue due to the organization of a former subarachnoid hemorrhage. At the end of the operation, the cortex pulsated almost normally. Usual closure with two drains of rubber tissue inserted. While the incision was being sutured, a lengthening of the left Achilles tendon was performed and, in this manner, the period of anesthesia was not prolonged. Duration, 45 minutes.

Post-operative Notes.—Uneventful operative recovery, so that the patient was discharged on the thirteenth day after operation; the decompression area bulged rather tensely, but slight pulsation was palpable; even at this early date, there seemed to be a definite lessening of the spasticity of the left arm and of the left leg; no convulsive seizures since the operation. Parents were advised to continue the massage and exercises of the left arm and left leg just as during the 3 years before the operation.

Examination (May 17, 1917—21 months after operation).—A marked improvement of the condition of this patient has steadily occurred since the operation, in that the left spastic hemiplegia has so lessened that the patient can walk with scarcely any limp of the left leg and she can use the left arm with much freedom and with only slight awkwardness—especially for the finer use of the fingers; the mentality has greatly improved, so that her school reports are much better than formerly and she has advanced one year in her classes; the convulsive seizures have lessened both in frequency and in severity—the first convulsion occurring 6 weeks after the operation and of comparatively slight character, and these modified spells have occurred on the average of only one every six or seven weeks; emotionally, the patient is much better in that she is less irritable and according to the mother "is a changed girl." The decompression area is slightly depressed beneath the flush of the scalp and pulsates normally; some new bone formation about the periphery has narrowed the opening slightly. Pupils equal and react normally. Reflexes: patellar—left very active; exhaustible left ankle clonus and left Babinski; abdominal reflexes —left depressed; deep reflexes of left arm more active than right. Fundi—

retinal veins slightly enlarged; no edematous blurring of the details of either optic disk but their new tissue formation and pallor still persist. Vision 16/20 in each eye.

Last Examination (July 16, 1919—47 months after operation).—The marked improvement of this patient has continued in that there is now only a very slight impairment of the left arm and leg, the mentality is becoming more and more normal in that the Simon-Binet tests register an age of 15 years and the convulsive seizures do not occur more frequently than once every two or three months. The decompression area is depressed beneath the flush of the scalp and pulsates normally. Reflexes: patellar—left more active than right; no ankle clonus but left Babinski persists; left abdominal reflexes depressed; reflexes of left arm more active than of right arm. Fundi—retinal veins possibly enlarged; no blurring of the margins of the optic disks disclosed. Vision still remains 16/20 in each eye.

Remarks.—The continuance of the convulsive seizures even in a less severe form and of less frequent occurrence makes the prognosis of this patient a most doubtful one ultimately and it would be most surprising if the epilepsy should entirely disappear: this is the type of patient and condition in whom a good result might possibly have been obtained, if the operative relief of the increased intracranial pressure could have been performed earlier—if not within several days after birth, then surely within the first two or three years of life. The end result of this patient will be reported later in detail.

It is most important in patients having convulsive seizures and there is present a definite increase of the intracranial pressure to ascertain accurately whether this increased intracranial pressure is a secondary one due to the cerebral edema which results from frequent convulsive seizures—the typical "wet brain" of epileptics, or whether the increased intracranial pressure is a possible factor in increasing the cortical irritability and therefore a primary rather than a secondary sign of the convulsions; it is only in these selected patients that the relief of the increased intracranial pressure affords a definite chance of improvement. An excellent method to ascertain whether the increased intracranial pressure is a primary rather than a secondary one following the convulsions is to saturate the patient with triple bromides or luminal sufficiently so that no convulsive seizures occur for a period of at least 4 weeks, and better, 6 weeks; if at the end of this period of freedom from convulsions, the pressure of the cerebrospinal fluid as registered by the spinal manometer is normal or only slightly above normal, whereas before this period of freedom from convulsions, the pressure was registered as being definitely increased (above 15 mm.), then it is known that the increased intracranial pressure is due to the convulsions themselves and results from the cerebral edema secondary to the convulsive seizures; whereas, if the pressure of the cerebrospinal fluid remains the same after the period of freedom from attacks just as before this period, then it is known that the increased intracranial pressure is a primary rather than a secondary factor and that a lowering of this increased intracranial pressure by means of a subtemporal decompression and drainage might afford the patient a definite chance of improvement by lowering the cortical irritability. This method of differentiating patients having convulsive seizures has been used in a large number of patients during the past two years and it has been most satisfactory; naturally, patients having convulsive seizures must be given most doubtful prognoses—no matter what the treatment, and especially if the condition has persisted for a period of years, so that the

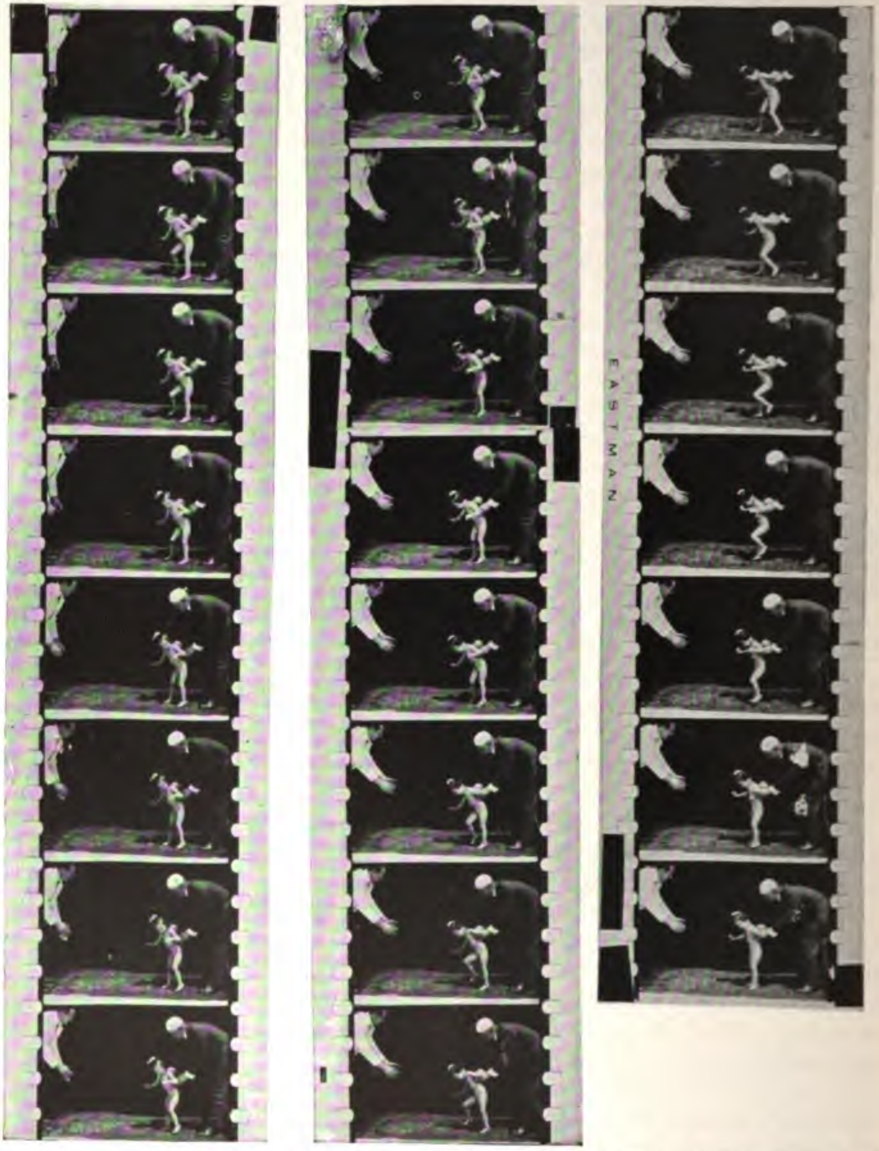


Fig. 204.—Severe condition of spastic diplegia with mental retardation in a child of 4 years of age following a supracortical and cortical hemorrhage at the time of instrumental delivery. Marked improvement following a lowering of the increased intracranial pressure by means of a left subtemporal decompression and drainage.

cerebral cortex has reached a degree of irritability which no treatment of any kind can permanently alter—the so-called epileptic habit. It is only in the very early conditions that it is possible to offer the patient a chance of recovery—and usually it is only a temporary improvement, if any at all.

CASE 181.—Chronic severe brain injury associated with cortical and supracortical hemorrhages and a resulting spastic diplegia and retarded mentality; a marked increase of the intracranial pressure. Left subtemporal decompression. Marked improvement.

No. 296.—Marjorie. Four years. White. U. S.

Admitted June 1, 1916—4 years after birth and injury. Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Operation June 7, 1916. Left subtemporal decompression and drainage.

Discharged April 16, 1916—9 days after operation.

Family history negative; no other children; no miscarriages. Parents and relatives are all right-handed.

Personal History.—First child, full term, head presentation, instrumental delivery; ten minutes required to resuscitate the child; weight, ten pounds; no convulsions. Child was considered a normal child until 6 months of age, when it was noticed that the right side of the body was slightly stiff and was not being moved as freely as the left side; child not holding its head up as early as it should normally. At 8 months of age, it was noticed that both legs were slightly stiff and spastic and there was a tendency to adduct the thighs—the right more than the left; it was also definitely ascertained that the right arm could not be moved as freely as the left arm. At 2 years of age, the child had improved sufficiently to be able to sit alone but was unable to stand alone; both legs were distinctly spastic with marked adduction; the speech was also retarded in that the child could say only "mamma," "papa" and several other short words. No convulsions at any time. The patient has received daily massage ever since the age of 6 months.

Examination upon admission (48 months after birth and cranial injury). —Temperature, 98.6°; pulse, 86; respiration, 26. Rather well-developed and nourished child. Head of normal size and shape. Mentality retarded in that child was not interested in its surroundings and less alert than normally; she says several short words but no sentences. Child cannot stand alone. (Fig. 204.) Both legs stiff with moderate flexion and adduction at knees; tendency to stand upon the toes of each foot—right more than left, due to mild Achilles contracture. Right arm slightly impaired in that the use of it was more awkward than of the left arm. Hearing negative (watch test). Pupils equal and react normally. Reflexes—patellar exaggerated, right more than left; right ankle clonus and double Babinski, Oppenheim and Gordon reflexes more marked on the right side; abdominal reflexes depressed; deep reflexes of the right arm slightly increased. Fundi—retinal veins enlarged; nasal margins of both optic disks blurred by edema; no obscuration of the other details of either optic disk. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 18 mm.); Wassermann test negative and cell count 3 cells per c.mm. X-ray (Doctor A. J. Quimby)—"convolutional markings of the entire vault and indicating an increase of the intracranial pressure" (Fig. 205).

Treatment.—In the belief that a lowering of this increased intracranial pressure by means of a subtemporal decompression, and if necessary, a bilateral decompression would afford this patient a lessening of the spasticity and a chance for a definite mental improvement so that the ultimate result would not be a hopeless condition, it was decided to perform a left subtemporal decompression as the right leg and the right arm were more impaired than the left arm and the left leg.

Operation (48 months after birth and injury).—Left subtemporal decompression: usual vertical incision, bone removed, and no complications. Dura whitish, thickened and tense; upon incising it, clear cerebrospinal fluid welled out, revealing a very "wet," edematous cortex which bulged and tended to protrude but did not rupture. Beneath the fissure of Sylvius, the cortex was negative except for the edema and a slight cloudy induration about the vessels in the sulci, but above the Sylvian fissure there was a bluish cystic formation lying in the cortex itself and extending upward beyond the bony margin of the decompression opening; it was almost 4 cm. in width. Upon incising its outer wall, a small amount of straw-colored fluid escaped, permitting the walls of the cyst to collapse and the underlying cortex to rise slightly, although it was very much atrophied. The surrounding cortex was negative except for the edema and the whitish cloudiness about the vessels in the sulci (the residue from the absorption of a former subarachnoid hemorrhage). As a profuse escape of cerebrospinal fluid had occurred, together with the evacuation of the cortical cyst, the cortex became less tense, and at the end of the operation it pulsated normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

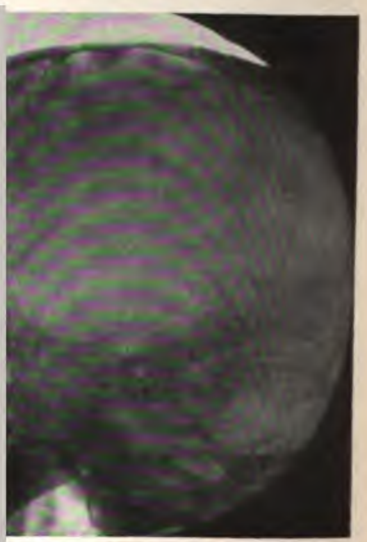


FIG. 205.—Lateral röntgenogram of a four year old child having the condition of spastic diplegia due to a supracortical hemorrhage at birth. The convolutional markings of pressure atrophy of the inner table of the vault indicate the prolonged increase of the intracranial pressure which was confirmed by the special tests.

Post-operative Notes.—Uneventful recovery; incision healed *per primam;* at discharge, the operative area bulged slightly beyond the flush of s pulsated normally.

Examination (May 20, 1917—11 months after operation).— marked improvement of both the spasticity and mentality has although the right arm and the right leg are still slightly stiffer th left side, yet the child is now able to walk with difficulty and th is very markedly lessened; mentally, the child is much brighter an ing in sentences. No convulsive seizures have occurred. Decompression area is slightly depressed beneath the flush of scalp. Reflexes—patellar active, right more than left; no ankle clonus and a left Babinski cannot be obtained, while a right Babinski only with difficulty and no Oppenheim or Gordon reflexes; deep reflexes of right arm possibly more active than of left. Fundi—retinal veins possibly enlarged; no edematous blurring of the nasal margins of either optic disk.

Last Examination (April 4, 1919—34 months after operation).—This patient has continued to improve so that she is able to walk and to run with only a slight spasticity of the right leg and an awkwardness in the use of the right arm. The mentality has so improved that the Simon-Binet tests would indicate the child as being of five years rather than six years of age. No convulsive seizures have occurred. Decompression area depressed; its size is slightly smaller owing to new bone formation about the periphery; pulsation normal. Reflexes—patellar, increased, right more than left; no ankle clonus but right Babinski still persists; deep reflexes of right arm possibly greater than those of left arm. Fundi—retinal veins negative; details of both optic disks clear and distinct. Parents were advised to continue massage and exercises and to prevent the child from leading a too exciting life at school—possibly on half time; no meats or meat soups, and no tea or coffee: to avoid, if possible, the extreme heat of the summer.

Remarks.—The marked improvement occurring in this patient, and especially following the operative findings of apparently a cortical hemorrhage and therefore with a permanent destruction of the affected cortical cells, is most gratifying; undoubtedly, the cortical cells beneath the cystic formation were compressed and functionally impaired rather than actually destroyed and atrophied, and this would account for the excellent recovery of function of the extremities and the lessening of the spasticity; the persistence, however, of a mild impairment of the right leg and less so of the right arm results most probably from a permanent damage of a number of the cells and this condition will undoubtedly continue.

The marked improvement of the mentality emphasizes the effect of a definite increase of the general intracranial pressure upon the development of children and the necessity of the early relief of this increased intracranial pressure is urgently indicated; naturally, the earlier the relief is possible the greater the recovery and the more normal the individual—the ideal time for the operative procedure being within several days after birth, when the hemorrhage itself can be drained and not merely the effects of the former hemorrhage as in these chronic patients years later.

The danger of convulsive seizures occurring in this patient and in others similarly affected is a great one and must always be feared; the chances of their occurrence in this patient are much less following the operation than if no relief of the increased intracranial pressure had been afforded.

CASE 182.—Chronic severe brain injury at birth associated with a supracortical and cortical hemorrhage resulting in a left spastic hemiplegia, mental retardation and convulsive seizures; an increased intracranial pressure. Right subtemporal decompression and drainage and one month later, a left subtemporal decompression and drainage. Marked improvement.

No. 48.—William. Eight years. White. Special school. U. S.

Admitted January 2, 1914—8 years after birth and injury. Hospital for the Ruptured and Crippled. Referred by Doctor Virgil P. Gibney.

Operations.—First, January 5, 1914; right subtemporal decompression and drainage. Second, January 31, 1914; left subtemporal decompression and drainage.

Discharged February 21, 1914—21 days after second operation.

Family history negative; two younger children living and well; no miscarriages for mother.

Personal History.—First child, full term, difficult labor requiring instruments; child remained in a semiconscious condition for one week, but no convulsive seizures occurred. Within one month after birth, the baby was considered a normal one and it was not until 8 months of age that it was observed that the left arm and left leg were slightly stiffer than the right arm and right leg; the child, however, was able to hold its head up at the normal age, but the time of its sitting up and attempting to walk was delayed several months. At 2 years of age, the child was able to walk with difficulty by dragging the left leg, which had become markedly spastic, adducted at the thigh and with a definite contracture of the left Achilles tendon, so that the child walked upon the toes of the left foot; the left arm was held flexed at the elbow and at the wrist with the hand pronated. The parents were instructed regarding massage and exercises of the left arm and left leg and these were administered daily up to the present time. At three years of age, the child "fainted"—suddenly stared, and fell from his chair, but did not "shake"; during the past five years, these "fainting" spells have occurred at irregular intervals of three months on an average and on only 6 occasions did the child have a convulsive seizure of the left side of the body—the attack always beginning in the left leg. The mentality was definitely impaired, so that the child is now going to a "special" school; very irritable—having "fits of temper." Patient has received the usual routine treatment of patients of this character—daily massage, exercises, etc.

Examination upon admission (8 years after birth and injury).—Temperature, 98.6°; pulse, 80; respiration, 26. Fairly well nourished. Typical condition of left spastic hemiplegia with flexor contractures of the arm and leg—the gait being a typical one with a dragging of the left foot and walking upon the toes of the left foot; left side of face smaller than the right side and definitely weak—being of the cortical type of left facial paralysis in that the left forehead muscles are not involved. Superficial Simon-Binet tests register the mentality as being about 5 years of age; rather dull and nonobservant of surroundings; very irritable. Hearing negative; otoscopic
examination negative. Pupils equal and react to light normally.
patellar—left very much exaggerated; left patellar and ankle cl
Babinski; abdominal reflexes depressed but equal; deep reflex
exaggerated. Fundi—retinal veins dilated; nasal mar
disks blurred by edema; small amount of new tiss
margins of both optic disks. Vision could not
to the lack of concentration of child. Lum

fluid under increased pressure (approximately 20 mm.); Wassermann test negative. X-ray negative.

Treatment.—This marked condition of left spastic hemiplegia with mental retardation and epileptiform seizures associated with an increased intracranial pressure was considered as being due most probably to an intracranial hemorrhage at the time of the birth of the child, and it was considered advisable to lower this increased intracranial pressure even at this late date of several years following the original injury; whether the increased intracranial pressure was due to hemorrhage or to the condition of external hydrocephalus or to some other cause unknown, yet the therapeutic indication was to relieve this increased pressure in the hope that the condition might then be improved—especially the lessening of the mental impairment and of the spastic hemiplegia, and, most important, to afford the patient an opportunity of relief from the epileptiform spells; for these reasons, a right subtemporal decompression and drainage was performed.

Operations.—First, right subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications; bone itself was unusually thick (almost 1 cm.), very spongy and vascular. (It has been ascertained in later patients having this condition of the overlying bone, that there is usually a large supracortical or cortical hemorrhagic cyst beneath this area of vascular and thickened bone—and it so proved in this patient.) Dura whitish, thickened, vascular and under high tension; upon incising it, much cerebrospinal fluid escaped, exposing a large supracortical and cortical hemorrhagic cyst—bluish in appearance and almost 2 cm. in thickness, lying almost entirely above the Sylvian fissure and extending upward beyond the upper margin of the dural opening; its outer wall was excised, allowing more than an ounce of straw-colored fluid to escape, and the walls of the cyst then collapsed. The underlying cortex had apparently been badly damaged in that it was very much atrophied and anemic; except for the cloudy induration about the vessels in the sulci, the surrounding convolutions were normal in appearance but under high tension. At the end of the operation, the cortex bulged but pulsated only feebly. Usual closure with 2 drains of rubber tissue inserted. Duration, 45 minutes.

Post-operative Notes.—Uneventful operative recovery; the decompression area, however, bulged so tensely and remained protruded for a period of over 3 weeks following the operation and with such slight pulsation, that it was considered wiser to lower this high intracranial pressure still more by means of a bilateral subtemporal decompression and drainage which was now performed.

Second Operation (left subtemporal decompression and drainage—26 days after first operation).—Usual vertical incision, bone removed, and no complications; the bone itself was of normal thickness and character. Dura whitish and tense, and upon incising it a small amount of cerebrospinal fluid welled out, revealing an edematous cortex; above the Sylvian fissure was a supracortical cystic formation resulting from a former subarachnoid hemorrhage; the underlying cerebral convolutions were only slightly compressed and apparently not damaged anatomically. About the vessels in however, was a small amount of whitish induration but of less extent than over the right cerebral cortex. At the end of the operation the cortex receded and pulsated normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Uneventful operative recovery, the incision healed *per primam* and at the time of discharge, on the twenty-first day after the second operation, both decompression areas bulged slightly beyond the flush of the scalp but pulsated normally; even before his discharge from the hospital, there was a definite lessening of the spasticity of the left arm and of the left leg, and it was possible for the patient to stand upon the left heel when pressing down upon the toes; the left arm was also much more "limber." The parents were advised to continue the massage and exercises as before the operations.

Examination (April 13, 1916—27 months after operations).—A marked improvement has steadily progressed during the past 2 years—both mentally and physically, and most important of all, there has been no recurrence of the epileptiform spells, although he "fainted" twice (the last time, one year ago) but he did not "shake." There is still a definite limp of the left leg and the left arm is used awkwardly, but he is now able to walk upon the left heel and to use his left arm, although not so well as the right arm (Fig. 206). The mentality has so improved that he is now able to go to school in a regular class but is two grades below the class for his age; not so irritable as formerly and as his father says, "a changed boy in every way." Reflexes: patellar—left more active than right; exhaustible left ankle clonus and double Babinski; reflexes of left arm more active than of right. Fundi—retinal veins slightly enlarged; no edematous blurring of the details of either optic disk; connective-tissue formation persists as before the operation. Both decompression areas are depressed and pulsate normally; slightly smaller due to new bone formation about the periphery.

Last Report (Sept. 22, 1919—67 months after operations).—Letter from father states, "William has steadily improved, and, although he is not yet a normal boy, he is developing mentally and physically each year. No fit during the past two years."

Remarks.—The operative findings of not only a supracortical but also a cortical hemorrhagic cyst of the right hemisphere indicate that the cortical damage over this area is a permanent one and therefore no complete recovery of the left arm and left leg is possible; however, an improvement was possible following the puncture of the cyst itself and the lowering of the general intracranial pressure owing to the fact that the cortical cells about the periphery of the cystic formation were merely compressed and functionally impaired—not destroyed as were probably the cortical cells lying directly beneath the cyst. A much greater improvement of the left arm and left leg would have been possible if the hemorrhage had bee
cortical one entirely rather than some of it being in the cortex its

The presence of the bilateral Babinski is explained by the
findings over the left cerebral hemisphere and if the hemorrhag
left cerebral hemisphere had been of larger amount, then undou
condition of this patient would have been one of spastic diplegia
rather than that of left hemiplegia alone.

If the intracranial pressure is so high that a unilateral decompression and drainage is not sufficient to lower the intracranial pressure so that the operative area becomes depressed beneath the flush of the surrounding scalp,

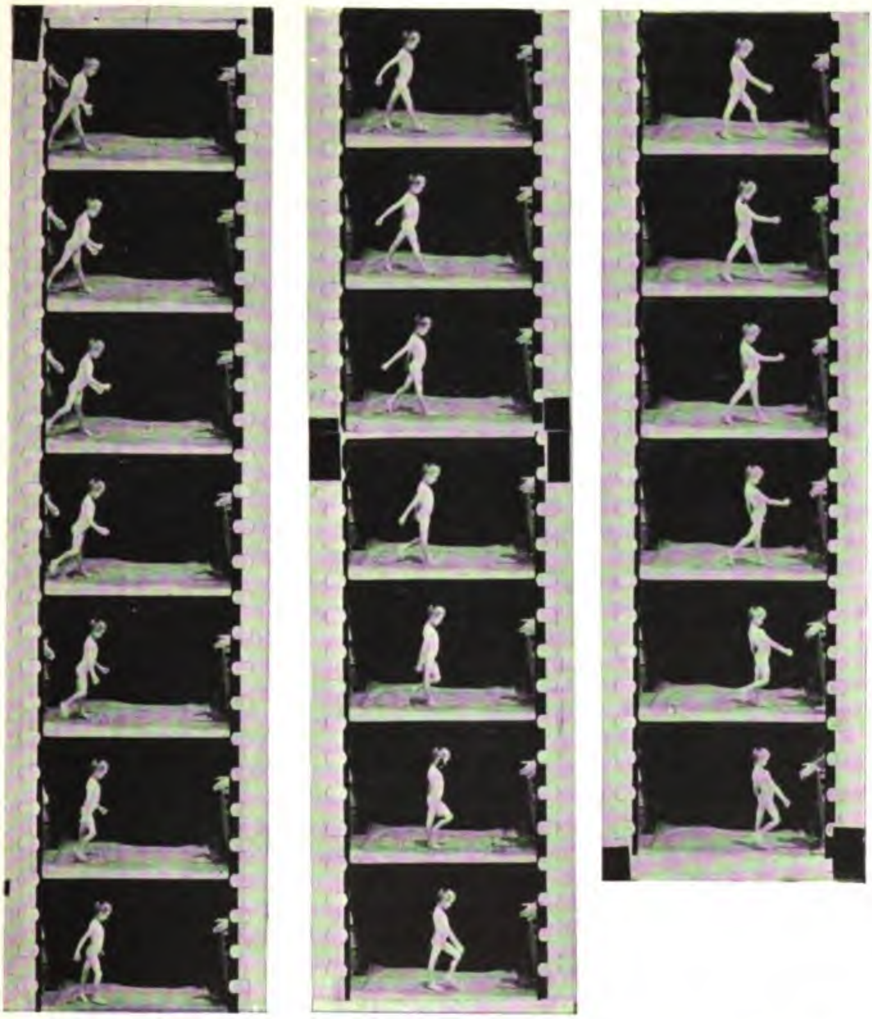


FIG. 206.—Two years following a lowering of the increased intracranial pressure in a boy of 8 years of age having the condition of left spastic hemiplegia with mental retardation and convulsive seizures; marked improvement.

then a bilateral decompression is always advisable and within a period of three months, or at most six months, following the first operation. The two operations should rarely be performed at the same time and only when the intracranial pressure is so high that the closure of the unilateral decompression would be a difficult procedure with possible damage to the underlying cortex, unless a bilateral decompression is immediately performed; this necessity rarely occurs.

CASE 183.—Chronic severe brain injury at birth associated with a supracortical hemorrhage and with a resulting condition of spastic diplegia, mental retardation and convulsive seizures; an increased intracranial pressure. Bilateral decompression and drainage. Marked improvement.

No. 14.—George. Ten years. White. U. S.

Admitted September 22, 1913—10 years after birth and injury. Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Operations.—*First,* October 3, 1913; right subtemporal decompression and drainage. *Second,* December 2, 1913—2 months after first operation; left subtemporal decompression and drainage.

Discharged December 12, 1913—10 days after second operation.

Family history negative; no other children; no miscarriages for mother.

Personal History.—First child, full term, difficult labor requiring instruments; child bluish for several hours after birth. One hour after birth, baby had a general convulsive twitching of both arms and of both legs and this continued each day for several minutes during the following week. Great difficulty in swallowing during this period. The convulsive seizures now ceased and the condition of the child so improved that he was considered a normal child at one month of age, and it was not until 7 months of age that it was observed that both arms and both legs were slightly stiff and that they were not being used freely. This stiffness and spasticity of the arms and legs gradually increased; a definite adductor contraction of the thighs was noticed at one year of age and a double Achilles contracture at 15 months of age. The general development of the child was also delayed in that he could not hold up his head until one year of age, could not sit up alone until 2 years of age, and was unable to stand until 4 years of age, but never alone and always requiring support. During this time, a "fainting" spell occurred on the average of one each month, but not always associated by a "shaking" of the arms and legs; during the past 4 years, there have occurred only 3 general convulsive seizures—the last one being 2 months ago. The stiffness and spasticity of the arms and legs, associated with contractures at the knees, ankles, elbows and wrists, gradually increased during these years, so that the child was unable to stand alone until 6 years of age and was first able to walk alone and with great difficulty and awkwardness only at 8 years of age (2 years ago); the knees are closely adducted in the "scissor" type of gait and both heels are so elevated by the extreme Achilles contractures that the child can only stand and walk upon the toes. The mentality has been greatly retarded. Speech has never been intelligent except to mother, who also complains of the extreme irritability of the child.

Examination upon admission (10 years after birth and injury).—Temperature, 98.6°; pulse, 78; respiration, 24. Rather poorly nourished. A typical condition of spastic diplegia with flexor contractures at both knees and ankles and both arms flexed at the elbows and wrists with the hands in the position of pronation; healed "sores" on the inner sides of both knees due to their rubbing in attempting to walk, owing to the marked adductor spasm of both thighs. Neither heel can touch the ground, as the contracture of both Achilles tendons is marked. Neither wrist can be extended nor the arms straightened at the elbows on account of the contractures of the flexor muscles. The use of the hands is very limited and the movements of the arms very uncertain and awkward. The patient has the greatest difficulty in walking—waddling in a jerky manner and most uncertain, the patient attempting to hold to any support. Frequent facial movements of an athetoid character with continuous drooling from the mouth. Mentality markedly retarded—Simon-Binet tests indicate an age of about 6 years; the unintelligible speech, however, and the great awkwardness of the hands interfered very much with the accuracy of the mental tests. Much difficulty in walking—waddling in a jerky manner and most uncertain, the unstable emotionally—has fits of temper and "tantrums." Hearing negative; otoscopic examination negative. Pupils equal and react to light normally. Reflexes—patellar very much exaggerated but equal; double patellar and ankle clonus; double Babinski, Oppenheim and Gordon reflexes; abdominal reflexes depressed but equal; deep reflexes of both arms equally exaggerated. Fundi—retinal veins dilated and their walls thickened with new tissue formation; nasal halves and temporal margins of both optic disks blurred by edema; much new tissue formation, causing both disks to be slightly paler than normally—a mild condition of secondary optic atrophy. Vision 14/20 in each eye. Lumbar puncture: clear cerebrospinal fluid under increased pressure (approximately 24 mm.); Wassermann test negative. X-ray (Doctor A. J. Quimby)—"signs of an increased intracranial pressure in the convolutional thinning of the vault, especially in the frontal and occipital areas" (Fig. 207).

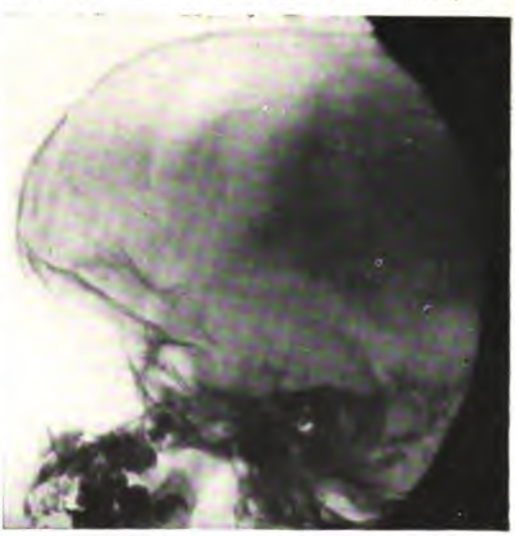


FIG. 207.—Convolutional markings of pressure atrophy of the inner table of the vault in a boy of ten years having a prolonged increase of the intracranial pressure due to a supracortical hemorrhage at birth; marked improvement following the operation of bilateral decompression and drainage.

Treatment.—The presence of a high intracranial pressure sufficient to produce secondary changes of the optic nerve disks—whether the intracranial lesion can be localized or not and whether the diagnosis is one of intracranial tumor, hydrocephalus, hemorrhage or what not—the therapeutic indication is to lessen this increased intracranial pressure, either by means of medicine or by the operation of cranial decompression; medicine being of no real value in these patients, it was decided to afford this patient a chance of improvement by means of a subtemporal decompression and drainage. The clinical history of a difficult labor immediately followed by convulsive twitchings and then later by an increasing spasticity of both arms and legs associated with mental retardation, occasional epileptiform spells and emotional instability and the persistent increased intracranial pressure—these facts would indicate a condition of supracortical hemorrhage at the time of birth and later, the development of an external hydrocephalus due to the partial blockage of the stomata of exit in the cortical veins of the cerebrospinal fluid, so that a severe condition of cortical edema is present. The lowering of this increased intracranial pressure by means of a subtemporal decompression and drainage and, if necessary, a bilateral subtemporal decompression and drainage is essential in order that an improvement be possible.

Operations (First, October 3, 1913—10 years after birth and injury).—Right subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications, except for the thinness of the bone. Dura thickened, whitish and under high tension; upon incising it, much cerebrospinal fluid welled through the dural opening, exposing a very edematous "wet" cortex; above the Sylvian fissure was a definite supracortical cystic formation of a former subarachnoid hemorrhage—about 1 cm. in thickness; upon puncturing its outer wall, a small amount of straw-colored fluid escaped from it. About the vessels in the sulci, both above and below the Sylvian fissure, was a whitish induration—most probably the remnant of the subarachnoid hemorrhage collected in the sulci and there becoming organized. At the end of the operation, the brain pulsated slightly and the cortical protrusion lessened, so that it was possible to approximate the edges of the overlying temporal muscle without damage to the cortex. Usual closure with 2 drains of rubber tissue inserted. Duration, 50 minutes.

Post-operative Notes.—Uneventful operative recovery; incision healed *per primam,* so that patient could be discharged on the eighth day after the operation; the decompression area bulged tensely but pulsated slightly and it was hoped that a bilateral decompression would not be necessary. This patient was repeatedly examined during the following 2 months and although a definite improvement occurred in the lessening of the spasticity of both the arms and legs, yet the decompression area bulged so tensely as a result of the intracranial pressure, that it was deemed advisable to perform a left subtemporal decompression in order to lower this pressure still more.

Second Operation (December 2, 1913—2 months after first operation).—Left subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications; the bone, however, was very thin—not much thicker than paper (due to pressure atrophy). Dura (as at first operation) was whitish, thickened and tense, and upon incising it a large quantity of cerebrospinal fluid gushed through the dural opening; upon enlarging the dural opening, a supracortical cystic formation extended upward beyond the Sylvian fissure and the upper edge of the dural opening as at the first operation; extensive cloudy induration about the vessels in the sulci. At first, the cerebral cortex protruded, but as the escape of much cerebrospinal fluid continued, this bulging lessened and the cerebral pulsation became almost normal. Usual closure with 2 drains of rubber tissue inserted. Duration, 45 minutes.

Post-operative Notes.—Uneventful operative recovery in that the incision healed *per primam* and it was possible for the patient to be discharged on the tenth day after operation. The parents were advised to continue the massage and exercises just as before the operations—also the advisability of this patient attending a "special" school.

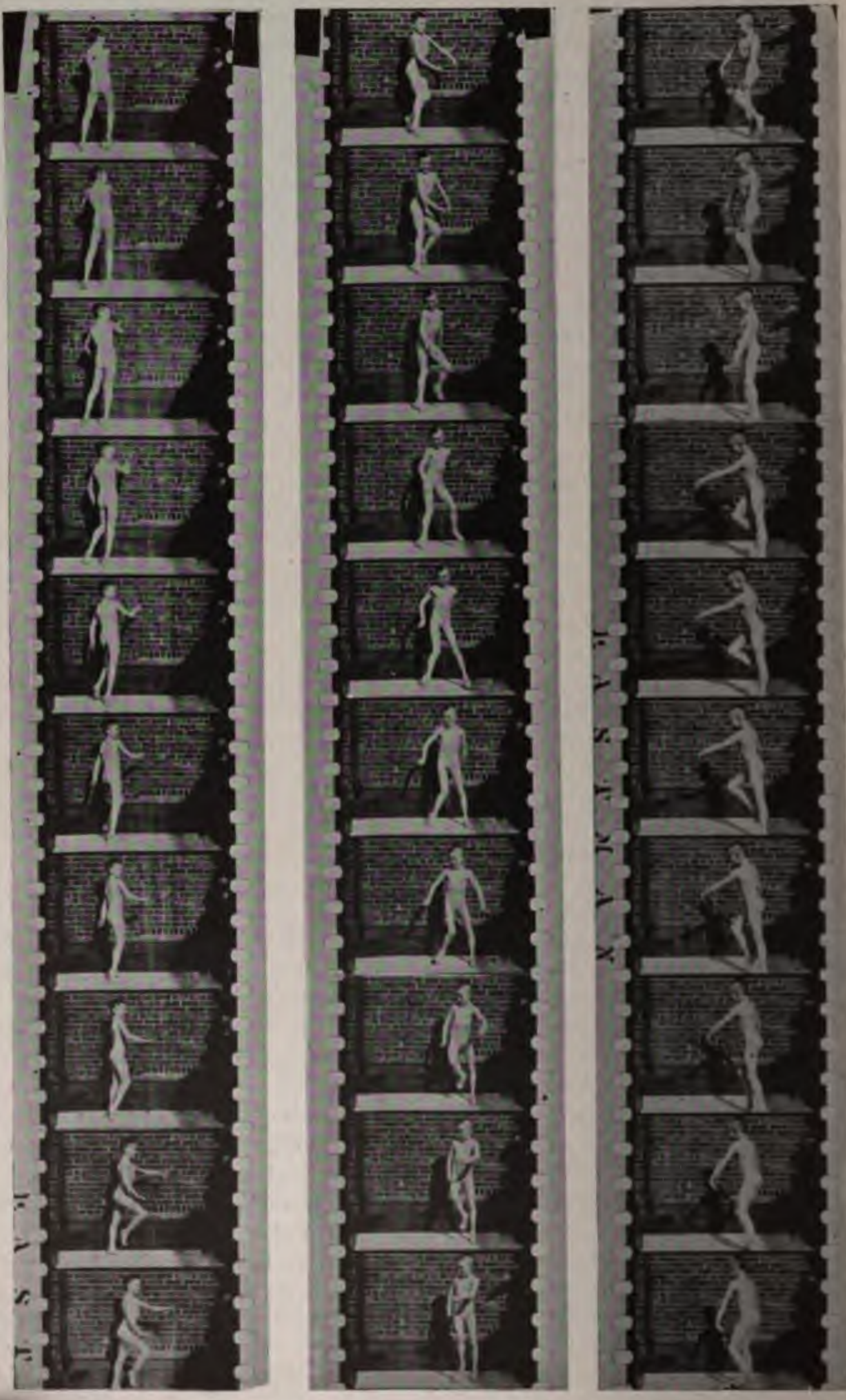
Examination (April 13, 1913—16 months after second operation).—The marked physical and mental improvement of this patient is very striking; not only has the spasticity lessened to such a degree that the patient can now walk easily although awkwardly, but a much freer use of the hands has resulted and the mental change is so marked that his teachers in school are most enthusiastic regarding his progress. It is now possible to understand his speech and he is so much more stable emotionally that he can be controlled without difficulty and his "fits" of temper are of very rare occurrence. No "fainting" spells or convulsive seizures have occurred since the first operation. Drooling from the mouth only occasionally occurs and only when the patient becomes excited. Both decompression areas bulge slightly beyond the flush of scalp; pulsation normal. Pupils equal and react normally. Reflexes—patellar active but equal; exhaustible right ankle clonus and double Babinski; deep reflexes of both arms active but equal. Fundi—retinal veins enlarged; lower nasal margins of both optic disks slightly blurred by edema; the secondary new tissue formation persists as before the operations. It is now possible to flex both feet dorsally to a right angle, so that both heels can touch the floor; adductor spasm of both thighs has lessened very much but it is still present; both arms can now be straightened at the elbow and the flexor contractures at both wrists are greatly improved (Fig. 208). The continuance of the routine daily massage and exercises is advised as before the operation.

Last Report (May 20, 1919—16 years after birth and 65 months after operation).

Patient has continued to improve in every way—especially mentally and physically; speech is now intelligible although the words are slurred at times, and no convulsive seizures have occurred. "In every way, George is more like a natural boy."

Remarks.—The continued improvement of this patient is most gratifying and especially when the condition is remembered as being such an extreme one of spastic diplegia—the greatest difficulty in walking alone, the restricted use of the hands, the retarded mentality, the unintelligible speech and the emotional instability and occasional epileptiform seizures—this condition in a boy of ten years of age, and, within a period of 6 years following the operations and a lowering of the increased intracranial pressure, the condition of this patient is so improved both physically and mentally that he no longer seems to be the same boy. He now walks alone to school where his mental progress has been most rapid; he is able to use both hands more freely and writes fairly easily; his speech is intelligible to all and he is emotionally normal as other boys of his age—and most important—the absence of convulsive seizures. All of this improvement has followed since the lowering of the increased intracranial pressure by means of the two subtemporal decompressions.

The presence still of a slight increase of the intracranial pressure could



g. 208.—Marked improvement of the condition of spastic diplegia in a boy of 10 years of age following a bilateral decompression and drainage to lower the high intracranial pressure.

now undoubtedly be avoided in similar patients by the use of the linen strands for a permanent and more complete drainage of the partially blocked cerebrospinal fluid—the condition in so many of these patients being one really of external hydrocephalus resulting from the partial blockage of the excretion cerebrospinal fluid through the stomata of exit in the walls of the cortical veins in the sulci; at present, all of these patients having very "wet," edematous brains are treated by this means of drainage thus making possible the lowering of the increased intracranial pressure to normal and the probability of its remaining normal.

The pressure atrophy of the bones of the vault to the extent of thinning them to the thickness of only a sheet of paper is quite common in these older patients in whom the intracranial pressure is high and prolonged over a period of years; röntgenograms are important in these patients in confirming the signs of increased intracranial pressure as revealed by the ophthalmoscope and the spinal mercurial manometer; negative röntgenograms, however, do not indicate the absence of an increased intracranial pressure.

Case 184.—Chronic severe brain injury occurring at birth and associated with a supracortical hemorrhage and a resulting spastic diplegia; an increased intracranial pressure. Bilateral subtemporal decompression. Marked improvement.

No. 545.—John. Sixteen years. White. School. U. S.

Admitted April 16, 1916—16 years after birth and injury. Polyclinic Hospital. Referred by Doctor John A. Bodine.

Operations.—First, April 23, 1916; second, May 3, 1917 (12 months later). Left and right subtemporal decompressions and drainage.

Discharged May 16, 1917—13 days after second operation.

Family history negative; four sisters living and well; father and mother living and well and no history of nervous disease in the families of either parents.

Personal History.—Third child, full term, difficult labor requiring instruments; "blue baby," becoming normal in appearance in three days; no cranial injuries noted; no convulsive seizures. Some difficulty in nursing the child in that there was, apparently, difficulty in swallowing and much regurgitation. Child was considered, however, a normal child until the eighth month, when it was noticed that both legs were slightly stiff—right one more than left, and also the arms possibly stiffer than usual. Child did not progress normally in that he was unable to hold up his head until the fifteenth month, could not sit alone until the twenty-sixth month and the stiffness of the arms and legs became more marked as the child became older; speech was also delayed until thirty months of age and even then only monosyllables were used and with much slurring and indistinctness; at 3 years of age, the incoördination of movements became more marked and although the child learned to walk at forty-six months of age, yet he did so with great difficulty and awkwardness, having a typical spastic gait associated with much muscular incoördination; athetoid movements now became more noticeable. The right arm and the right leg were always stiffer and more awkward than the left arm and the left leg—the right foot dragging. The condition was diagnosed as "Little's disease" and the routine treatment of massage and exercises advised but the prognosis was considered absolutely bad; no ophthalmoscopic examinations or lumbar punctures were performed to estimate accurately the presence or not of an increased intracranial pressure and thus the condition was not differentiated from those patients having a spastic paralysis due to a lack of development and naturally not having an increased intracranial pressure. During the past few years, the condition of spastic paralysis of both arms and of both legs has persisted together with the marked impairment of speech, so much so that the patient has great difficulty in making himself understood—the mentality being only slightly impaired. No convulsive seizures have occurred.

Examination upon admission (16 years after injury).—Temperature, 98.6°; pulse, 80; respiration, 24; blood-pressure, 118. Fairly well-developed and nourished. Marked spastic paralysis of both arms and legs—the latter more affected; both legs are adducted so that the knees tend to cross; legs slightly flexed at the knees and a definite talipes equinus is present—right more than left; he walks with much difficulty and awkwardness, dragging the right foot. Both arms stiff but less so than the legs, and no contractures; right arm more impaired than the left; on attempting to use the hands, the coarse tremor and incoördination increase. The speech is markedly impaired in that all of the words are slurred and indistinct, and it is with difficulty that he can be understood. Marked impairment in swallowing so that occasionally liquids return by the nose. Mentality slightly impaired—the Simon-Binet tests registering an age of 12 years plus rather than the actual age of 16 years. The emotional reactions are fairly normal. Pupils: equal and react to light and accommodation normally; no nystagmus; no ocular paralyses. Reflexes: patellar—both exaggerated, right more than left; inexhaustible patellar and ankle clonus; bilateral Babinski, Oppenheim and Gordon reflexes; abdominal reflexes present and equal; deep reflexes of both arms and masseteric reflexes increased. Fundi—retinal veins slightly enlarged with their walls thickened; both optic disks possibly paler than normally, with a definite edematous obscuration of the nasal margins; both physiological cups shallow from connective tissue formation. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 16 mm.); Wassermann test negative and cell count was 4 cells per c.mm. X-ray (Doctor W. H. Stewart)—"negative."

Treatment.—The history and the presence of an increased intracranial pressure, associated with a spastic diplegia, more on the right side than on the left side and with only slight mental impairment, made advisable the operation of left subtemporal decompression to lower this increased pressure in the belief that a definite improvement of this patient would res at this late date, following the original intracranial injury; the of convulsive seizures was also an encouraging prognostic factor in the operation advisable.

Operation (16 years after injury).—First, left subtemporal de sion and drainage: usual vertical incision, bone removed, and n cations. Dura thickened, whitish and under high tension; upon i clear cerebrospinal fluid spurted to a height of 3 inches, and upon enlarging the dural opening a very "wet," edematous cortex tended to protrude but did not rupture, owing to the rapid escape of much cerebrospinal fluid. Below the fissure of Sylvius, the cortex was apparently normal, but extending down to the Sylvian fissure was a cystic supracortical formation lying beneath the arachnoid and filled with a straw-colored fluid; upon excising its outer wall and allowing this fluid to escape, the underlying compressed cortex was enabled to rise after the cyst itself collapsed; several of the underlying cerebral convolutions appeared pale and atrophied from pressure; this cystic formation was at least 1 cm. in thickness and extended upward over the parietal lobe as far as could be seen beyond the bony edge of the decompression opening. About the vessels in the Sylvian fissure was a whitish induration due to the organization of the former hemorrhage about the vessels, and this same condition was present about the vessels in the sulci. (Apparently the cystic formation seen at operation was the lower portion of an extensive hemorrhagic supracortical cyst resulting from the former intracranial hemorrhage at the time of birth and as both sides of the patient's body are affected, it is most probable that the cortex of the other hemisphere is also similarly impaired.) At the end of the operation, the cortex pulsated almost normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 35 minutes.

Post-operative Notes.—Uneventful convalescence; incision healed *per primam* and the patient was discharged on the twelfth day; the decompression area bulged slightly but pulsated almost normally, and it was decided to wait for a period of 6 months to 1 year in the hope that the increased intracranial pressure would be entirely relieved by this one operation, as would be indicated by the usual tests of intracranial pressure and the "sinking-in" and depression at the site of the decompression opening; the routine treatment of massage, exercises, etc., was advised as before.

During the following year, the patient made a definite improvement in that the spasticity of both arms and legs lessened, but more of the right side of the body than of the left, so that the patient now could use the right arm and right leg better than the left arm and left leg. (This is an interesting observation and of frequent occurrence in these patients—a left decompression causing a more marked improvement and lessened spasticity of the right side of the body than of the left side and *vice versa,* and this is what one would naturally expect.) The speech had improved so that it was more intelligible and his school teacher reported a greater mental capacity and aptitude. The decompression area, however, continued to remain flush with the surrounding scalp and it did not become depressed so that a bilateral decompression was considered advisable.

Examination upon second admission (April 26, 1917—17 years after the injury and one year after operation).—Temperature, 98.6°; pulse, 80; respiration, 24; blood-pressure, 120. Definite spastic diplegia but much less than one year ago; the stiffness and awkwardness of the left arm and left leg are now greater than that of the right arm and right leg; the right leg no longer drags and patient walks less upon the toes than before. The speech has markedly improved and he can be understood with less difficulty;

no impairment of swallowing. Mentality has definitely improved—Simon-Binet tests now registering him as of the age of almost 15 years; his emotional control is also better. Pupils present and react to light and accommodation. Reflexes—patellar very active, left possibly more than right; exhaustible right patellar and ankle clonus, but inexhaustible left patellar and ankle clonus; double Babinski, Gordon and Oppenheim reflexes; abdominal reflexes present and equal; deep reflexes of both arms increased—left possibly more than right. Fundi—retinal veins enlarged and a definite blurring of the nasal margins of both optic disks—left possibly more than right; otherwise the fundi are the same as at the preceding examinations of one year ago. Lumbar puncture—clear cerebrospinal fluid under increased pressure (13 mm.). The site of the former decompression over the left side of the head pulsates but it is not depressed and remains flush with the surrounding scalp; at times, it bulges.

Treatment.—The increased intracranial pressure having been lessened but not sufficiently to permit the greatest ultimate improvement, it was considered advisable to perform a right subtemporal decompression in the belief that a greater lessening of the pressure would make possible a still greater improvement.

Second Operation (17 years after injury and one year after first operation).—Right subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications. Dura thickened, whitish and tense; upon incising it, clear cerebrospinal fluid spurted to a height of one inch and upon enlarging the dural opening the underlying edematous cortex bulged but did not rupture. No definite fibrous or cystic formation ascertained, but about the vessels in the sulci was a cloudy whitish thickening of connective tissue—the organization of the former supracortical hemorrhage which could not be entirely absorbed and lying in the sulci. (It is the blockage in this manner of the stomata of exit of the cerebrospinal fluid in the walls of these cortical veins in the sulci which causes the "wet," edematous condition of the brain—in reality, a mild condition of external hydrocephalus; the more complete this blockage, the greater is this cortical edema and excess cerebrospinal fluid.) So much cerebrospinal fluid escaped that by the end of the operation, the cortex pulsated normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Uneventful convalescence; incision healed *per primam* and the patient was discharged on the thirteenth day. At discharge, the decompression areas were both slightly depressed beneath the flush of scalp. The usual routine treatment of these patients of massage, exercises and general hygiene was advised—just as before the operation.

Examination (January 29, 1918—9 months after second operation).—
The improvement of the patient has rapidly progressed in that the spas
of the arms and legs has markedly lessened so that now the patient ca
both hands and can walk with much less difficulty and awkwardness
ever before; he is able to ride a bicycle to school and his speech
improved that it is possible for him to be in the classes with the othe
he has even played baseball, but naturally with difficulty. A defi
provement mentally has also occurred and he is now more interested

and desires to learn. Not so irritable and "gets along" with the other boys much better. Both decompression areas are depressed and pulsate normally. Reflexes—patellar less active than before, right being possibly greater than left; exhaustible patellar and ankle clonus; double Babinski reflexes persist; deep reflexes of both arms increased but much less than before. Fundi—retinal veins possibly slightly enlarged but no edematous blurring of the nasal margin of either optic disk can be observed; the pallor of both disks naturally persists due to the former scar tissue formation. Visual acuity 16/20 in each eye—the same as before the operations. No lumbar puncture was performed as both decompression areas were depressed and therefore no marked increase of the intracranial pressure could be present.

Last Report (March 6, 1919—23 months after last operation).—Mother writes: "Jack has continued to improve during the past year, both in his physical condition—walking particularly—and also in his school work. The places where the operations were made are sunken but their beating is still visible. He does not complain of headaches and his entire condition is most pleasing."

Remarks.—It is to be regretted that this patient could not have been operated upon earlier—at the time of birth or surely as soon as the results of the intracranial condition first appeared—within one year after birth; if such a marked improvement can be obtained sixteen and seventeen years after the cranial injury, surely the earlier operation would not only prevent the greater physical impairment, but it would have afforded the patient the opportunity of developing more normally. It is most surprising that this patient has improved so much as has occurred since the operation, and it merely emphasizes again that the intracranial lesion in this patient and in the vast majority of patients similarly impaired and having an increased intracranial pressure, that there is not a primary destruction of the cerebral cortex but that the function of the cortical cells is impaired by the increased intracranial pressure resulting both from the supracortical hemorrhage and the partial blockage of the excretion of the cerebrospinal fluid, producing in most of these patients a mild condition of external hydrocephalus. By lowering this increased intracranial pressure by means of a decompression and, if necessary, a bilateral decompression, then the cortical cells are no longer compressed to the extent as before and therefore they are now able to functionate more normally.

One of the earliest signs of improvement following the successful lowering of this increased intracranial pressure is the lessening of the spasticity and awkwardness of the affected arm or leg, a greater ease of speech and most fortunately an improved mentality, so that these patients come out of their "haze," become more interested in their surroundings and are thus enabled to develop mentally; their emotional control becomes greater and therefore they are more stable.

Naturally, in these older patients it has never been suggested or intimated that their condition can be cured, so that they would become normal just as if the condition had not happened; they can be improved, however, and in the milder patients the condition can be so benefited that they approximate normality, but the older they become without a lowering of this increased intracranial pressure, just so much less can be expected of a later relief of the increased intracranial pressure. The younger the patient, the greater chance of affording the greatest ultimate improvement (the intracranial lesion being the same), and naturally the ideal time for the operation is within several days after birth when the free supracortical hemorrhage can be immediately drained in fluid form, and in this manner not only is the increased intracranial pressure immediately lowered, but the secondary complication of a blockage of the excretion of the cerebrospinal fluid is thus anticipated and prevented. If in all doubtful cases following birth—with and without the use of instruments—and the child is abnormally quiet or surely in the presence of convulsive twitchings, if a lumbar puncture is then performed and an increased pressure ascertained and especially in the presence of blood in the cerebrospinal fluid, then an immediate drainage of the subdural blood by means of repeated lumbar punctures and spinal drainage or by a modified subtemporal decompression operation would afford these unfortunate children their opportunity for the greatest ultimate recovery—both of life and of normality.

CASE 185.—Chronic severe brain injury at birth associated with an extensive supracortical hemorrhage and a resulting spastic paraplegia and mental retardation; an increased intracranial pressure. Bilateral subtemporal decompression. Marked improvement.

No. 19.—Mary. Twelve years. White. Special school. U. S.

Admitted October 2, 1913—12 years after birth and injury. Orthopedic Hospital. Referred by Doctor B. P. Farrell.

Operation October 14, 1913. Bilateral subtemporal decompression and drainage.

Discharged November 6, 1913—22 days after operation.

Family history negative.

Personal History.—First child, full term, normal labor apparently—no instruments being required; no convulsive seizures. Bottle baby. The child was considered normal in every way until the eleventh month, when it was noticed that both legs were slightly stiff, and this spasticity gradually increased during the next six months until the legs became adducted and flexed at the knee, together with marked contraction of both Achilles tendons; daily massage and exercises were given both in the hospital and at home, and yet the spasticity gradually became more and more marked. No definite impairment of the arms observed. The child has never been able to walk or to stand alone. During the past eight years, three operations for lengthening both Achilles tendons have been performed with a resulting improvement during the following 6 to 8 months, when the contractures gradually returned as before. Mentality has become definitely impaired but it is comparatively slight compared with the extensive spastic paralysis of
No convulsive seizures at any time. Patient has received excelle
treatment of massage and exercises daily, but no permanent i
has been obtained.

Examination upon admission (12 years after birth and
perature, 98.6°; pulse, 82; respiration, 26. Well-nourish
marked spastic paraplegia associated with flexor contr

at the hips and at the knees, and a bilateral plantar flexion due to the marked contraction of both Achilles tendons; child stands when supported upon the toes with the "scissor" type of adduction of both thighs. Both arms are used comparatively freely and with little or no impairment. Patient talks with only a slight retardation and difficulty. A cursory Simon-Binet test registers an age of nine years. Hearing negative; otoscopic examination negative. Pupils equal and react normally. Reflexes—patellar very much exaggerated but equal; double patellar and ankle clonus; double Babinski, Gordon and Oppenheim reflexes; abdominal reflexes depressed but equal; deep reflexes of both arms—possibly more active than normally. Fundi—retinal veins dilated; nasal margins of both optic disks blurred by edema; new tissue formation about the margins of both optic disks which are possibly paler than normally—a mild secondary optic atrophy. Vision 18/20 in both eyes. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 18 mm.); Wassermann test negative. X-ray report—"negative."

Treatment.—The presence of the increased intracranial pressure, as indicated by the ophthalmoscope and lumbar puncture findings, makes advisable its operative relief by means of a subtemporal decompression, in the belief that its lowering would permit a lessening of the spasticity, an improvement of the mentality and the greatest ultimate recovery of function in this patient—in spite of the advanced age and the severity of the spastic paraplegia to the extent of an inability to stand alone or to walk; the danger of future convulsive seizures is also very great indeed.

Operations (12 years after birth and injury).—Bilateral subtemporal decompression and drainage. First, right decompression: usual vertical incision, bone removed, and no complications. Dura whitish, thickened and tense; upon incising it, clear cerebrospinal fluid spurted through dural opening and upon excising its outer wall, a small amount of straw-colored fluid escaped, permitting the underlying edematous cortex to protrude. Supracortical vessels were very much dilated and about them in the sulci was a whitish induration, especially about those above the Sylvian fissure, where the arachnoid was very much thickened and of a hazy appearance—as of a cyst formation; upon puncturing it, however, clear cerebrospinal fluid escaped. The cerebral convolutions appeared possibly paler than normally. Owing to the continued protrusion of the cerebral cortex, it was feared that a rupture of it might occur unless a left subtemporal decompression was immediately performed and this was done before the closure of the right decompression was attempted.

Second Operation.—Left decompression: usual vertical incision, bone removed, and no complications. Dura thickened, whitish and almost as tense as at first operation; upon incising dura, clear cerebrospinal fluid welled out under tension, revealing a definite cystic formation above the Sylvian fissure and upon excising its outer wall, a small amount of straw-colored fluid escaped, permitting the underlying convolutions to rise. Much whitish induration about the vessels in the sulci. At the end of the operation, the cortex pulsated almost normally due to the escape of much cerebrospinal fluid. Usual closure of both decompression openings with 2 drains of rubber tissue inserted. Duration, 75 minutes.

Post-operative Notes.—Uneventful operative recovery; both incisions healed *per primam;* even at discharge upon the twenty-second day after operation, there was a definite lessening of the spasticity and a brighter, more alert mental condition. The usual routine treatment of massage and exercises was continued just as before the operation.

Examination (April 13, 1915—18 months after operation).—An excellent improvement has occurred in that the patient was able to walk with support within 8 months after the operation and she now walks alone and erect with only a slight "waddle"; a brace devised by Doctor Farrell has been most effective in aiding the walking. Mentality is much improved and the child has now advanced to a "special" sixth grade. Much better control emotionally. Decompression areas protrude slightly beyond the flush of scalp; normal pulsation. Reflexes—patellar very active but equal; no patellar nor ankle clonus, but double Babinski reflexes persist; abdominal reflexes present and equal; deep reflexes of both arms negative. Fundi—retinal veins enlarged; lower nasal margins of both optic disks slightly blurred by edema; new tissue formation remains as before the operations. Vision—18/20 in each eye.

Last Report—January 20, 1919—63 months after operations. Report of district nurse: "Mary has continued to improve in her walking but she still uses braces; she can move about more freely. She is very bright in her mind." A report of the condition of this patient will be made in detail later—at the end of a ten-year period.

Remarks.—The excellent improvement occurring in this patient, even at the late age of 12 years following a lowering of the increased intracranial pressure due to a partial blockage of the excretion of the cerebrospinal fluid by means of a former supracortical hemorrhage with new tissue formation about the supracortical veins and the presence of a supracortical hemorrhagic cyst itself—this marked improvement, both physically and mentally, has been a most gratifying one. Although this patient is not a normal child in that the walking is definitely impaired and awkward, yet she is able to walk and her mental improvement has been so striking that it is not only a most encouraging result but a cause of great regret that the operative lessening of the increased intracranial pressure could not have been performed years earlier and in this manner a much greater ultimate recovery of function would have been possible; as is now well realized, the ideal treatment is within several days after birth, when the supracortical hemorrhage can be itself drained rather than at a later operation to lessen the effects and results of this supracortical hemorrhage—the partial blockage of the excretion of the cerebrospinal fluid into the cortical veins with the f
of a mild condition of external hydrocephalus. It is most difficul
ceive that any brain injury can occur, and especially if its effects
are prolonged, that the patient can become, no matter what th
a perfectly normal individual—mentally, emotionally and
only in the early patients—as soon as possible followi
lesion, that it is possible for these patients to approxi

medical efforts should be directed toward the early diagnosis of the lesion and its appropriate treatment.

The persistence of a slight bulging of both operative areas indicates an increase of the intracranial pressure, and that in this patient both decompressions and drainage were not sufficient to decrease the intracranial pressure down to its normal amount. To avoid this complication, it has been the practice in our clinic during the past two years to treat these patients just as are the patients having the condition of external hydrocephalus—that is, through the decompression opening, several linen strands are inserted subdurally and beneath the arachnoid and are brought out through the temporal muscle and fascia into the subcutaneous tissues of the scalp in a stellate manner, and thus it is believed that a permanent drainage of the partially blocked cerebrospinal fluid is possible by means of the numerous lymphatics of the scalp; at least, since using this latter method of operative procedure and drainage, it has been possible to lower the intracranial pressure to normal in these patients so that no protrusion of the decompression areas has occurred.

The absence of convulsive seizures in this patient, and especially in view of the operative findings of the supracortical hemorrhagic cyst and the new tissue formation about the cortical veins in the sulci—a sufficient cortical irritant to produce convulsive seizures in most patients—is surprising and yet a very common observation in almost one-half of these patients. Why they do not have convulsions and yet practically the same intracranial lesion is present, may possibly be due to a greater nerve stability—whether due to ancestry or to some other factor. Naturally, the prognosis is much more favorable in the patients who have not had convulsions or at least not since early childhood than in the ones in whom convulsions are of frequent occurrence.

Case 186.—Chronic cranial injury occurring at the time of birth and followed by convulsive twitchings, spastic diplegia and an increasing intracranial pressure. Right subtemporal decompression and drainage of large brain abscess. Death. Autopsy.

No. 1043.—Elizabeth. Twelve weeks. White. U. S.

Admitted November 20, 1918—12 weeks after birth and injury. Audubon Hospital. Referred by Doctor M. H. Bass.

Operation.—November 27, 1918. Right subtemporal decompression and drainage of cerebral abscess.

Discharged December 3, 1918—6 days after operation.

Died April 17, 1919—6 months after birth and 3 months after operation. Acute internal hydrocephalus.

Family history negative; no other children; no miscarriages for mother.

Personal History.—First child, full term, difficult instrumental labor (high forceps); much difficulty in resuscitating the child. Three days after birth, a temperature of 103° developed and it was noticed that both arms and both legs could not be moved; this continued for 4 weeks, when it was observed that both arms and both legs were becoming stiff, the head was held backward, the neck slightly stiff and a forward arching of the back (a mild condition of opisthotonos); spasmodic twitchings now appeared in the left side of the face, left arm and left leg, lasting for several minutes and occurring as frequently as 20 times each day; a general convulsive seizure resulted about every three days. During the past 2 months, these epileptiform spells have continued together with an increasing spasticity of both arms and legs and a definite enlargement of the head; child has not gained in weight and has become very much emaciated.

Examination upon admission (12 weeks after birth and cranial injury).—Temperature, 101.2°; pulse, 124; respiration, 34. Very much emaciated; skin wrinkled, presenting a senile appearance; weight, 12 pounds. Body rigid—back arched forward and head held rigidly backward. Child does not notice anything—eyes rolling from side to side. Severe spasticity of both arms and legs, especially the left side. Veins of scalp are enlarged and very extensive. Head is larger than normal with a definite protrusion of both fontanelles, which are very tense, and a marked widening and separation of the suture lines, especially the mid-frontal suture. Pupils of normal size and reaction to light. Reflexes—patellar, very much exaggerated, left more than right; no patellar or ankle clonus, but a left Babinski obtained; abdominal reflexes absent; deep reflexes of both arms increased —left more than right. Fundi: retinal veins enlarged; distinct edematous blurring of the nasal halves and the nasal margins of both optic disks which are rather white. Lumbar puncture: three attempts were made to obtain some cerebrospinal fluid for examination and to measure accurately its pressure, but no fluid could be obtained—only a small quantity of blood: (this inability to obtain the cerebrospinal fluid at repeated lumbar puncture was undoubtedly due to a blockage of the cerebrospinal fluid within the cranial cavity, so that it was not possible for the fluid to descend into the spinal canal—that is, either the condition of adhesions about the foramen magnum or the condition of an internal hydrocephalus). X-ray report —"a distinct overlapping of the bones forming the lambdoidal suture and a possible fracture of the occipital bone itself" (Fig. 209).

Treatment.—The history of instrumental delivery to be followed within 3 days by an increased temperature and weakness of the extremities and then 4 weeks later, by a spastic paralysis of both arms and legs, epileptiform spells of a localized character and then finally by the enlargement of the head with protruding tense fontanelles and a separation of the lines of suture and the presence of a high intracranial pressure being confirmed by the ophthalmoscopic examination, and the X-ray findings of cranial trauma in the occipital area—these data all tended to indicate the cranial injury at the time of birth followed by a meningeal inflammation (a mild meningitis) with the later development of an internal hydrocephalus which prevented the cerebrospinal fluid from descending into the spinal canal. This was the tentative diagnosis and the operation of right subtemporal decompression and drainage was advised in the belief that the institution of satisfactory drainage might prove of benefit to the condition; the operation was advised as the only known means of improving the condition by a drainage of the internal hydrocephalus.

Operation (12 weeks after birth and cranial injury).—Right subtemporal decompression and drainage of a brain abscess; usual vertical incision.

bone removed and no complications; no Doyen perforator or bu
as the bone itself was of a membranous character and it was po
all of these babies, to incise the pericranium between the squam
parietal bones and thus rongeur away the surrounding bone to
of 2 inches. Dura thickened and tense so that it bulged; upon
no cerebrospinal fluid escaped, and in order to lessen the high c
sion for fear that the underlying cortex would rupture, an attem
to tap the right ventricle by means of the ventricular punc
Upon inserting the puncture needle into the upper convolution
temporal lobe and at right angles to the cortex, when it had reached a depth of not more than 2 cm., a large amount of yellowish pus welled up through the needle and around it, and upon enlarging this cortical puncture opening, over 2 ounces of similar pus escaped. Pathological report (Doctor Jeffries): "Pure growth of a gram-negative bacillus." A rubber tube for drainage was now inserted into the abscess cavity which appeared to be about 3 inches in diameter and filling the right temporo-sphenoidal lobe and the lower portion of the right parietal lobe. The cerebral tension immediately lessened and no further attempt was made to tap the ventricle for fear of extending the infective process, and it was also now considered an unnecessary procedure as
the cerebral tension had been lowered by the drainage of the ab
Usual closure with 2 drains of rubber tissue inserted subdurally;
tube drainage of the abscess was naturally left *in situ*. Duration.

FIG. 209.—An overlapping of the bon
lambdoidal suture and a possible fracture of th
in a child following an instrumental deliver
spastic diplegia and convulsive seizures. A ri
decompression to lower the increased intracra
closed a large brain abscess. which was drai
position of the head due to the rigid posterior
neck to form with the arched spinal column
opisthotonos.)

Post-operative Notes.—An excellent operative recovery o
that the drainage of the abscess continued for 2 days
gradually ceased, so that it was possible to remove the dra
8 days after the operation, when the abscess cavity itself had
collapsed; the child gained 3 ounces in weight daily and al
convulsive twitchings of the left side of the body continued
days, yet they were much less severe and infrequent than before
tion and no general convulsive seizure occurred. The operati

healed and the general condition of the child had so improved, that it was possible for it to be discharged from the hospital on the sixth day after operation. During the following month, the general condition of the child improved and at times no convulsive twitchings of the left side of the body occurred for days; the operative area, however, began to bulge during the fourth week following the operation, and for fear that the abscess was being refilled with a purulent secretion, it was decided to explore through the operative incision; nothing abnormal was found, however, except an increased intracranial pressure, and it was considered to be better surgical judgment not to attempt a ventricular drainage for fear of extending the infective process. Following this exploratory incision, the general condition of the child improved for a period of 2 months, in that the spasticity became less severe, the epileptiform twitches less frequent and at times a normal intracranial pressure was disclosed by the ophthalmoscope; the child continued to gain in weight. Four months after operation, the condition rapidly became worse in that the size of the head enlarged, the fontanelles bulged tensely and the ophthalmoscope revealed an edematous blurring of the nasal halves of both optic disks; the child could no longer nurse and the condition itself rapidly became so much worse from the acute ventricular dilatation that it was decided not to attempt any surgical procedure of drainage owing to the extreme condition of emaciation and weakness of the child; the temperature gradually ascended to 106 plus, the pulse and respiration finally could not be counted and the child died from extreme weakness apparently and inanition, 6 months after birth and 3 months after the operation.

Autopsy.—Typical hydrocephalic head in the bulging forehead and occipital areas and the widely separated suture lines. No fracture of the cranial bones found. Operative site negative except for several adhesions between the temporal muscle and the underlying cerebral cortex; there were also numerous adhesions at the base, especially subtentorially about the foramen magnum. The cerebral convolutions were rather flattened as the result of the increased pressure, but otherwise the cortex itself was negative. In the right temporo-sphenoidal lobe was a healed abscess cavity of 3 inches in length and 1½ inches in width; it was well walled-off from the surrounding tissue. Both lateral ventricles and also the third ventricle were widely dilated and filled with a clear cerebrospinal fluid (bacteriological report, "no organisms found"). Both middle ears contained a purulent secretion.

Remarks.—Before this patient was operated upon, the diagnosis was one of an increased intracranial pressure due most probably to an internal hydrocephalus or to an extensive intracranial hemorrhage occurring at the time of birth and secondarily followed by a blockage of the cerebrospinal fluid. (The condition had been diagnosed at numerous consult
Little's disease due to a lack of cerebral development and agenes
cortex, although there were present at the time the signs of an i
intracranial pressure which naturally made these diagnoses unte
well as impossible.) The operation itself of decompression wa
simply to lower the increased intracranial pressure—whether
blockage of the ventricles themselves or to a former hemorrhag

tumor, or to any condition that would cause a marked increase of the intracranial pressure—the presence of an increased intracranial pressure being in itself sufficient justification and indication of the necessity of an operative lowering of it if the condition of the child was to be improved. The finding of a brain abscess at the operation was a surprise and it had not been diagnosed, but the operative indication was the same, no matter what the intracranial condition happened to be, the therapeutic object being to lower the increased intracranial pressure; in this case of a brain abscess, this lowering of the increased intracranial pressure was easily possible by means of drainage through the decompression opening; the risk of the serious complication of a meningitis following the drainage of a subcortical abscess is lessened by the drainage through a decompression opening by means of which the increased intracranial pressure is lessened and the surrounding tissues are more enabled to withstand an infective process.

It is very rare to be unable to obtain cerebrospinal fluid by means of a lumbar puncture—a so-called "dry" tap. It does occur most frequently, however, when the intracranial condition is due to an internal hydrocephalus and especially in the presence of an acute purulent meningitis, since in these conditions it is possible for the cerebrospinal fluid to be prevented from descending into the spinal canal as the result of adhesions and in the latter case from the purulent exudate itself. "Dry" taps, however, should never be accepted as such, until several attempts have been made to obtain the fluid at lumbar puncture and should only be so considered when it is definitely known that the needle is within the dural sac of the spinal canal. In this patient, the absence of cerebrospinal fluid at lumbar puncture was most probably due to adhesions about the foramen magnum and also to the presence later of a complete internal hydrocephalus. The röntgenogram disclosing the overlapping of the bones of the occipital area was a possible cause originally for the beginning of the infective process and the development of the cerebral abscess; this is to be doubted very much as there were no signs of an underlying meningitis having been present; in the presence, however, of a purulent secretion in both middle ears, the autopsy findings would tend to indicate this as being a possible channel for the source of the infection.

The excellent operative recovery following the drainage of the abscess itself was most gratifying and the prognosis was most encouraging following the continued post-operative improvement of the general condition of the baby; if an acute blockage of the ventricles had not occurred as the result of adhesions subtentorially, it might have been possible for an excellent improvement to have been obtained, although the end-result could not have been a satisfactory one, owing to the extensive damage to the brain itself and to the fact of the extreme severity and degree of the intracranial lesion.

Case 187.—Chronic severe brain injury at birth associated with a supracortical hemorrhage, producing the condition of severe spastic diplegia, mental impairment and convulsive seizures; an increased intracranial pressure. Bilateral decompression. Two weeks later, at operation to lengthen the Achilles tendons, death from ether. Autopsy.

No. 515.—Edward. Fourteen years. White. Spain.

Admitted February 15, 1915—14 years after birth and injury. Polyclinic Hospital. Referred by Sir Victor Horsley.

Operations.—Bilateral decompression, March 3 and 15, 1915; tendon lengthening, March 31, 1915.

Death March 31, 1915—immediately following the induction of ether narcosis for the operation of tendon lengthening.

Family history negative; no other children; no miscarriages for mother.

Personal History.—First baby; full term, prolonged "dry" labor; but no instruments; no convulsions after birth and the child was considered normal until 6 months of age, when it was noticed that the head was held slightly backward and rather stiff. At 7 months of age, it was observed that the left leg and the left arm were not being moved as freely as the right arm and leg and that the left leg was possibly stiffer than the right leg. At 8 months of age, both legs were observed to be stiffer than normally and one month later, a definite adduction appeared. Child was able to hold up its head at 12 months of age and he was able to sit up at 17 months of age. There was no attempt made to talk. The spasticity of both legs and of both arms—more on the left side—gradually increased until there developed the typical condition of spastic diplegia with adduction of both legs in the "scissor" type of posture and with flexor contractures of both arms and legs—always more on the left side. At 3 years of age, the first general convulsive seizure occurred with no localizing signs and lasting for a period of five minutes; the second seizure occurred 9 years later (2 years ago), and since then there has occurred each week 2 general convulsive seizures; no localizing signs, and usually at night. He has attempted to talk but cannot make himself understood. Mentality markedly impaired. Patient has never been able to stand alone or to walk. No improvement has resulted from daily massage and exercises.

Examination upon admission (14 years after birth and injury).—Temperature, 98.6°; pulse, 80; respiration, 24; blood-pressure, 114. Rather poorly nourished. Patient presents a typical picture of spastic diplegia associated with marked flexor contractures of both legs—the left more than the right; double talipes equinus with marked adductor spasm of both thighs; flexion of both arms at the elbows and at the wrists; both thighs flexed upon the trunk and the back is slightly arched. No rigidity of the neck. Patient is able to use both arms and legs in a stiff awkward manner—the left side being worse than the right. Unable to talk but he can make certain guttural sounds which can be differentiated by the parents as meaning "Yes," "No," "I want to," etc. Much difficulty in swallowing—fluids frequently returning through the nose. Apparently no impairment of hearing; otoscopic examination negative. Patient is able to stand upon the
toes, but he must hold to a chair or other object for support; atten
walk when supported, but the adduction of both thighs is so marked
has the greatest difficulty in moving one knee beyond the other; the l
is much worse than the right. Pupils equal and react normally.
bismus nor nystagmus. Reflexes—patellar exaggerated, left mo
right; double patellar and ankle clonus, but a Babinski reflex is c
only on the left foot; abdominal reflexes depressed but equal; deep

of both arms increased; masseteric reflexes increased equally. Fundi—retinal veins enlarged with thickened walls and much new tissue formation about them; nasal halves of both optic disks and also their temporal margins blurred by edema; both optic disks rather pale from new tissue formation, presenting an appearance of mild secondary optic atrophy; both physiological cups shallow with new tissue formation. Lumbar puncture—clear cerebrospinal fluid under high intracranial pressure (approximately 24 mm.); Wassermann test negative and the cell count was 5 cells per c.mm. X-ray (Doctor A. J. Quimby)—"pronounced convolutional markings of increased intracranial pressure."

Treatment.—The condition of this patient was such an extreme one and of such severity, especially in view of the advanced age of the patient, that it made the treatment of whatever character of so little value in that it could not be conceived that this patient could become normal or even approximate normality; that is, even with the most successful outcome, the most that could be expected would be an improvement—a lessening of the spasticity to the extent of possibly walking, an improved mentality and the ability to speak a few words intelligibly. This was fully explained to the parents but they felt that if any improvement at all could be offered by an operative relief of the high intracranial pressure, then it should be afforded to the child. Accordingly, the operation of right subtemporal decompression was performed in the belief that the lowering of the increased intracranial pressure would permit a definite improvement of the condition to occur.

Operations (14 years after birth and injury).—First, right subtemporal decompression: usual vertical incision, bone removed, and no complications: bone was unusually thick, vascular and spongy—being almost one-half of an inch in thickness along the anterior margin. Dura thickened, whitish and tense; upon incising it, clear cerebrospinal fluid spurted to a height of 4 inches, revealing a very "wet," edematous cortex beneath the Sylvian fissure, while above the Sylvian fissure was a bluish cystic hemorrhagic mass lying upon the cortex and possibly within the cortex itself, and extending upward beyond the bony edge of the decompression opening. The cortex bulged tensely and tended to protrude, but it did not rupture, as much cerebrospinal fluid continued to escape so that at the end of the operation the cortex pulsated feebly. A portion of the cystic formation became concave at each respiration, as if a cortical destruction had occurred at that point. The outer wall of the cyst itself was incised, permitting a straw-colored fluid to ooze out and causing the outer wall of the cyst to assume a concave position. The cortex beneath the Sylvian fissure was negative, except for the presence of the edema and a cloudy induration about the vessels in the sulci; the convolutions themselves were possibly paler and more anemic than normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 35 minutes.

Post-operative Notes.—Uneventful recovery in that the incision healed *per primam* and no complication occurred; the decompression area, however, protruded tensely beyond the flush of scalp so that 12 days later, it was considered advisable to perform a bilateral decompression.

Second Operation.—Left subtemporal decompression: usual vertical incision, bone removed, and no complications; bone itself was very much thickened, spongy and vascular—being similar to the bone removed at the first operation. Dura thickened, whitish and moderately tense; upon incising it, clear cerebrospinal fluid welled through dural opening, revealing a very edematous "wet" cortex but no cystic formation was exposed—only a whitish induration about the vessels in the sulci and a thickening of the arachnoid above the Sylvian fissure. At the end of the operation, the cortex bulged slightly but pulsated almost normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Uneventful recovery; incision healed *per primam.* Within one week, there was a definite lessening of the spasticity of both legs and of both arms and it did seem that the child was more alert mentally than before. Both decompression areas bulged beyond the flush of scalp but pulsated normally.

Examination at discharge (10 days after operation).—Temperature, 98.6°; pulse, 84; respiration, 24. Both decompression areas bulged slightly but with normal pulsation. Adductor spasm of both legs definitely lessened —possibly more in the left leg. The Achilles contracture of each foot is still present owing to the very much shortened tendon—really anatomically short owing to the prolonged contracture over a period of years, so that the feet could not be flexed dorsally to a position of right angles (just as before the operation). Swallowing is less difficult, however, and the speech is somewhat more intelligible, according to the parents but our examinations do not confirm it. The patient is able to shake hands much more easily and with less awkwardness than before the operation. Pupils equal and react to light normally. Reflexes—patellar exaggerated, left still greater than right; exhaustible patellar clonus and also exhaustible right ankle clonus, but an inexhaustible left ankle clonus still persists; left Babinski but right plantar flexion; deep reflexes of both arms less than before the operation. Fundi—retinal veins enlarged; edematous blurrng of nasal margins alone of both optic disks; the connective tissue formation about the optic disks and the retinal veins naturally persists, also the pallor of each disk.

Treatment.—On account of the double talipes equinus due to the shortened Achilles tendons, a double tendon lengthening was advised upon the return of the child to the hospital the following week; the patient was accordingly removed to a suburban home for a period of 5 days, when it was brought to the hospital for the operation of tendon lengthening. Upon the following day, the child having been prepared for the operation and being in a perfectly satisfactory condition, the usual administration of ether was begun and the child was just entering into the second stage of ether narcosis when respiration suddenly ceased; all attempts toward artificial respiration, the use of oxygen and the pulmotor were of no avail
cyanosis continued and finally the heart ceased to beat withi
of 8 minutes.

Autopsy.—Head: operative areas healed perfectly; the bone
was very much thickened, vascular and spongy and especiall
parietal areas, where it reached a thickness of one-half of an
beneath these areas there were the definite signs of a former su

hemorrhage; the dura was very much thickened and over the right parietal cortex was a supracortical cystic formation which had compressed and atrophied the underlying cortical cells; over the left parietal cortex, there was no such definite cystic formation—merely a thickening of the arachnoid and a whitish induration about the vessels in the sulci; this latter condition was present over the cortex of both hemispheres with the exception of the anterior portions of both frontal lobes and the posterior portions of both occipital lobes. The cystic formation over the right parietal cortex produced a slight concavity of the cortex itself, but there was apparently no primary destruction of the cortical cells at the time of the hemorrhage—that is, the condition was one of supracortical hemorrhage and the clinical impairment was due to compression of the underlying cortical cells and not to a primary destruction of them. Ventricles were negative. Heart and lungs negative. Kidneys negative. The ductless glands were apparently normal in size.

Remarks.—Although the ultimate result to be obtained in this patient could not be expected to be a very encouraigng one, yet it is most unfortunate that whatever improvement might have been obtained, even in a patient so badly impaired and at such a late age, that it was not at least afforded to this patient and to the parents—it being an only child. The findings at autopsy, however, are most instructive in that not only was the diagnosis of a supracortical hemorrhage confirmed, but it was demonstrated that an early drainage of this supracortical hemorrhage within a short time after the birth of the child would not only have lessened the future impairment, but it might have been possible to have secured a child who would approximate a condition of normality.

The cause of death could not be ascertained by the autopsy and it is possible that the third anesthetic within a period of one month had so lessened the resistance of the child that merely the anesthesia, for some reason yet unknown, should affect the vital centres in the medulla and thereby produce a cessation of the respiration and finally the heart. The value for having a permission for autopsy signed before operations is very well illustrated in this patient, in that not only is the diagnosis confirmed but the cause of death is at least known to be due to no condition which might have been ascertained before the operation and therefore corrected or the operation avoided; in many patients, however, the cause of death is ascertained and in this manner future patients can be spared a greater risk than otherwise would be possible.

Case 188.—Chronic severe brain injury at birth associated with a supracortical hemorrhage causing a marked mental retardation; high intracranial pressure. Right subtemporal decompression and drainage; 6 months later, left temporal decompression and drainage. Death. Autopsy; thrombosis of single lateral sinus.

No. 941.—Herbert. Six years. White. U. S.

Admitted December 6, 1917—6 years after birth and injury. Polyclinic Hospital. Referred by Doctor O. S. Hoffman, Omaha.

Operations.—First, January 16, 1918—right subtemporal decompression and drainage. Second, May 8, 1918—left subtemporal decompression and drainage.

Died May, 8, 1918—16 hours after second operation.

Family history negative; two younger children living and well.

Personal History.—First child, full term, difficult labor requiring instruments; head contused, especially the right side of forehead and over the right parietal eminence; difficult to resuscitate, and it was noticed that the child was unusually quiet during the first two weeks and had difficulty in nursing; no convulsive twitchings observed. Child was considered a normal baby until 4 months of age, when spasmodic momentary contractions of both arms occurred and later of both arms and legs; apparently no loss of consciousness until three months later, when these general convulsive seizures would occur four and five times each day accompanied with a rolling of the eyeballs and at times with involuntary urination and defecation; this continued until nine months of age, when they ceased for one month and then they returned much more frequently and of greater severity for three months (13 months of age); this convulsive condition now improved and gradually disappeared until the last spell, which occurred when the child was 33 months of age, and there has been none since, although there have always been much restlessness and excitability since that time. A general stiffness of mild degree of both arms and legs was observed when the child was 8 months of age and this spastic condition increased until 14 months of age, when there was a gradual lessening of it, so that at 2 years of age the child was able to stand alone, and at 3 years of age he was able to walk with only a slight awkwardness and no marked spasticity. No attempt has ever been made to talk; he understands, however, simple things; remembers tunes well—humming them months later. Patient runs and plays with the greatest abandon but he cannot concentrate upon anything for any length of time. The usual daily treatment of massage and mental training but with no improvement.

Examination upon admission (6 years after birth and injury).—Temperature, 98.6°; pulse, 84; respiration, 26; blood-pressure, 114. Well-developed and nourished. Head slightly larger than normal and the forehead tends to tower over the face, so that the eyes appear sunken and the face small in proportion to the rather large square head. No definite spasticity of the arms and legs ascertained; no adductor or flexor contractures and the child walks normally upon both heels and can run normally. Marked impairment of mentality in that he notices things only momentarily and apparently does not recognize relatives—only his nurse. Extremely restless—does not remain quiet more than momentarily—jumping about, clapping his hands and making guttural noises but no intelligible speech. Hearing negative; otoscopic examination negative. Patient can use both hands freely and without any awkwardness. Pupils equal and react normally. Reflexes—patellar exaggerated but equal; no ankle clonus but a suggestive right Babinski; abdominal reflexes depressed but equal; deep reflexes of both arms active but equal. Fundi (Doctor J. A. Kearney)—"retinal veins dilated, tortuous and buried in edematous tissue in places; general regressive edematous changes throughout the disks; nasal halves of both optic disks blurred by edema; the entire surface of the fundi around the disks presents a finely pepper-shaken appearance; in the equatorial region there is an irregular distribution of pigment in which the colloidal circulation may be seen in places." Lumbar puncture—clear cerebrospinal fluid under high intracranial pressure (30 mm.); Wassermann test negative and cell count was 4 cells per c.mm. X-ray (Doctor G. W. Welton)—"convolutional markings typical of an increased intracranial pressure; this convolutional pressure atrophy of the vault has greatly thinned the bone" (Fig. 210). Head measurements—hat circumference, 20½ inches; nasion-occipital protuberance, 13 inches; intermeatal, 15 inches.

Treatment.—It was most surprising to ascertain such an extremely high intracranial pressure and for fear that an error of technic had occurred, a lumbar puncture with a measurement of the pressure of the cerebrospinal fluid by means of the spinal mercurial manometer was again made one week later, and the pressure registered a height of 26 mm.; a third measurement of the pressure of the cerebrospinal fluid was taken one week later and a registration of a pressure of 24 mm. was obtained. (At each lumbar puncture, 6–8 c.c. of cerebral fluid were removed and this drainage possibly accounted for the slight lessening of the pressure; no anesthetic or local anesthesia was necessary for the puncture and the child remained perfectly quiet and relaxed at each puncture.) On account of this high intracranial pressure, as indicated by the lumbar punctures and by the ophthalmoscopic examinations, it was considered advisable to perform a right subtemporal decompression in the belief that a lowering of this increased intracranial pressure would afford this patient a definite mental improvement and the greatest ultimate recovery possible, as it was realized that a marked improvement of the condition of this child could not occur in the presence of such a high intracranial pressure, and that later even a greater impairment would result from the prolonged cerebral compression.

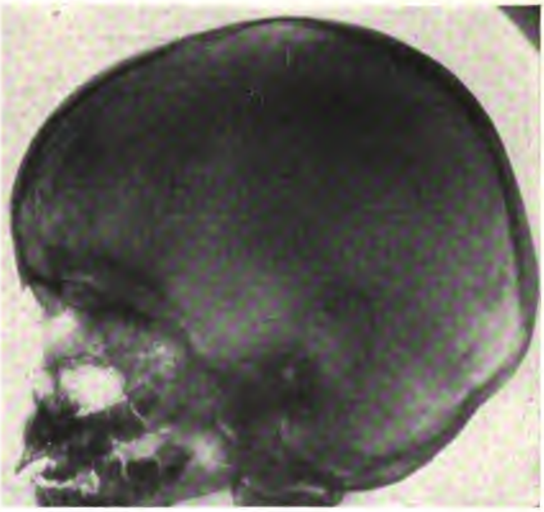


FIG. 210.—A prolonged increase of the intracranial pressure in a boy of 6 years of age producing pressure atrophy of the inner table of the vault, and chiefly in the frontal area—the so-called convolutional markings.

First Operation.—Right subtemporal decompression and drainage; usual vertical incision, bone removed, and no complications; the bone itself was unusually thin—in places being less than 1/16 and even 1/32 of an inch, almost like tissue paper (and due undoubtedly to the prolonged intracranial pressure). Dura thickened, whitish and very tense; upon incising it, clear cerebrospinal fluid spurted to a height of 3 inches, and upon enlarging the dural opening, the underlying edematous cortex protruded, but fortunately it did not rupture. (The pressure was similar to that as is frequently found in patients having cerebral tumors.) Above the Sylvian fissure was a supracortical cystic formation of almost 1 cm. in thickness, but it apparently extended forward toward the frontal lobe and backward to the occipital lobe and not upward over the motor areas of the parietal cortex. About the vessels in the sulci was a cloudy induration—undoubtedly due to the organization of a former free subarachnoid hemorrhage. At the end of the operation, the cortex bulged but pulsated slightly; much cerebrospinal fluid had escaped during the operation. The outer wall of the cystic formation was excised and four linen strands inserted and brought out through the open dura and the temporal muscle and inserted beneath the scalp in a stellate manner (just as in similar patients having a condition of mild external hydrocephalus secondary to a former supracortical hemorrhage or meningitis). Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

FIG. 211.—Definite improvement in a child of 6 years of age following a decompressive lowering and drainage of the increased intracranial pressure resulting from a supracortical birth hemorrhage and a congenital absence of the right lateral sinus.

Post-operative Notes.—Uneventful recovery; incision healed *per primam* and the patient was discharged on the sixteenth day after operation, at which time the decompression area bulged beyond the flush of scalp but pulsated slightly.

The patient was sent to Wildwood, Pa., to be under the charge of Doctor E. Bosworth McCready; during the next 5 months, a marked improvement occurred in that not only did the child become brighter mentally but the restlessness and ceaseless movements of the hands and legs became very much less, and he appeared to take more interest in his surroundings and toys (Fig. 211); no real attempt, however, was made to speak. On account of the persistent and tense bulging and protrusion of the decompression area, it was considered advisable to perform a left subtemporal decompression and drainage in order that this increased intracranial pressure might be lessened still more and permit a greater ultimate improvement; accordingly, the child was again returned to the hospital.

Examination (5 months after operation).—Temperature, 98.6°; pulse, 82; respiration, 24; blood-pressure, 116. Definite improvement in that the child is much quieter than before the operation, sits in a chair or upon the bed playing with his toys and is in every way much more interested in his surroundings and in the people who enter the room; sleeps well throughout the night. Pupils equal and react normally. Reflexes—patellar active but equal; no ankle clonus and no Babinski can be elicited; abdominal reflexes depressed but equal; deep reflexes of both arms—present and equal. Fundi (Doctor J. A. Kearney)—"retinal veins dilated but not buried in edematous retina; only the nasal margins of both optic disks obscured by edema; the other changes persist as at the former examination." Lumbar puncture—clear cerebrospinal fluid under increased pressure (16 mm.). The decompression area of the former operation bulged rather tensely beyond the flush of scalp and pulsated strongly.

Treatment.—Although the high intracranial pressure had been lowered from that of over 24 mm., to its present height of 16 mm., yet the persistence of this increased intracranial pressure as registered by the spinal mercurial manometer, the ophthalmoscope and by the continued bulging at the site of the former operation, it was decided to perform a left subtemporal decompression in the hope that a normal intracranial pressure might be secured and therefore the greatest ultimate recovery of this patient.

Second Operation (5 months after subtemporal decompression).—Left subtemporal decompression and drainage: usual vertical incision, bone removed, and no complications; as on the right side, the bone was exceedingly thin, being almost like tissue paper at its lower portion. Dura thickened, whitish and tense; upon incising it, clear cerebrospinal fluid welled through the dural opening, exposing a "wet," edematous cortex which protruded but did not rupture. No cystic formation observed, but about the vessels in the sulci was a whitish induration—the connective tissue of the organization of a former subarachnoid and supracortical hemorrhage. Much cerebrospinal fluid escaped, permitting the cortex to pulsate normally at the end of the operation. Four linen strands were inserted beneath the dura supracortically and brought through the temporal muscle and fascia and inserted under the scalp in a stellate manner as a means of permanent drainage. Usual closure with 2 drains of rubber tissue inserted. Duration, 35 minutes.

Post-operative Notes.—The child recovered from the anesthesia in good condition and except for an unusual amount of restlessness, nothing abnormal was noted. Twelve hours after operation, the temperature was 102°, pulse 88, respiration 28, and the child was sleeping quietly following the administration of codeine, grains $\frac{1}{4}$, hypodermically; the discharge of cerebrospinal fluid from the operative incision was not abnormally profuse and to all appearances, the condition of the child was excellent. Five hours later (15 hours after operation), the child continued in the same good condition; 30 minutes later, it was observed by the nurse that the child's face was slightly cyanosed although there was no cardiac or respiratory difficulty; upon summoning the house-surgeon, who arrived within 5 minutes, the face of the child had become very dark—conjunctival vessels also being dilated, and it was then observed that the respiration was shallow and slightly irregular; an attempt at artificial respiration was made but the condition of the child rapidly became worse, the face becoming practically black from the extreme cyanosis, the respiration first ceased and then the heart stopped beating—4 minutes later; that is, within a period of 15 minutes, the excellent condition of this patient rapidly changed to a most serious one, resulting in death—all within this short period of minutes.

Autopsy.—Extreme dilatation of all the vessels of the scalp. Both decompression areas negative. Upon removing the vault, it was observed that the dural venous emissaries were all enlarged and particularly the longitudinal sinus, which was dilated and bulging. Upon opening the dura, the cortical veins were widely dilated, giving the cortex a very bluish congested appearance; the cerebral hemispheres themselves were very edematous, "water-logged" and their venous channels all dilated. It was now ascertained that a left lateral sinus did not exist as the result of a congenital malformation and defect, and the right lateral sinus had enlarged to the size of one inch in diameter as compensation and in it was found a large thrombus almost 2 inches in length—completely occluding its lumen and thus being the cause for the sudden change in the child's condition. Even the pons and medulla were also very much congested and "water-logged" with edema. Over the cortex of both frontal lobes and over the right occipital lobe, was a cystic formation—the residue of a former supracortical hemorrhage; about the cortical vessels in the sulci was a whitish connective-tissue formation resulting from the organization of a former subarachnoid hemorrhage. Ventricles negative.

Remarks.—The importance of having obtained a permission, in writing, for an autopsy before the operation, is again emphasized in this most unfortunate death of a patient, for whom the greatest hopes were held of a marked future improvement; not only did the autopsy findings confirm the diagnosis of a former supracortical hemorrhage as being the primary cause of the intracranial impairment and the resulting mild external hydrocephalus being the secondary factor in continuing this increased intracranial pressure, but they disclosed the immediate cause of the patient's death. No cause for the congenital absence of the left lateral sinus could be ascertained as it is probable that this child could have gone through life with only one lateral sinus with its compensatory dilatation affording a sufficient channel for the venous return of the intracranial blood. The sinus being on the right side, if a thrombus in it were to form it is thought it would occur more probably following the right subtemporal decompression rather than following the left subtemporal decompression as it did in this patient. Naturally, once the thrombus in this single sinus occurred, the effect of its presence was so overwhelming to the patient that death resulted before it was possible for any treatment to be of value.

The absence of a definite cystic formation over the cortex
parietal motor area explains the absence of a marked spasticity in t
and the general increase of the intracranial pressure was the ca
increased reflexes but not to the extent of a definite spastic para
presence of the hemorrhagic residue, chiefly over the front
occipital lobes, produced the marked mental impairment as
only a slight physical impairment. It is most unfortunate th

could not have been afforded the opportunity of recovery by means of an early cranial drainage of the supracortical hemorrhage within several days after the child's birth and its occurrence, and in this manner the secondary effects of the partial blockage of the cortical veins could have been avoided so that there would not have developed the condition of mild external hydrocephalus. A most interesting case but a most unfortunate death.

B. *Chronic brain injuries occurring in children.*

The persistent effects of brain injuries occurring in children before the age of ten and twelve years are very similar to those occurring in adults with the important exception that since the mental and emotional "make-up" of the children is in the process of development, any prolonged impairment of function during this formative period is later exhibited in a greater retarded mental and physical condition; especially is an emotional instability to be feared, and if a definite cortical irritation is present to a degree that a mild chronic cerebral edema exists, then the danger of epileptiform seizures is one of not only great frequency but of the direst consequences to the patient; if once convulsions occur, and especially if months and years after the brain injury, then the chances of benefiting the patient are just that much lessened and the longer the convulsions persist the greater the improbability of any procedure being of any permanent assistance to the patient; on the contrary, epileptiform seizures occurring at the time of the acute intracranial condition or within a short period following it—these patients are frequently restored to a normal mental and emotional condition by the appropriate medical treatment.

The fact that children withstand the acute effects of brain injuries much more easily than do adults, and particularly is this true of the severe conditions of initial shock and high intracranial pressure, many children having brain injuries have been carelessly treated and the remote effects of the intracranial lesion have been overlooked—merely because the patient has made an immediate recovery of life; this latter result is all-important, but the future normality and the good health of the child should also be considered. It has been recognized for a number of years that all depressed fractures of the vault should be elevated or removed at the time of the acute injury—not only for the immediate benefit to the patient but to lessen the danger of future impairment and complications, and especially emotional instability and epileptiform seizures; this routine method of treatment has been advocated chiefly in children on account of their developmental period of life when an intracranial lesion, however insignificant its present symptoms and signs may be, yet its remote effects are frequently of a most serious character. The significance, however, of a persistent and chronic increase of the intracranial pressure following brain injuries in children, whose apparent excellent recovery from the immediate effects of the injury has been complete, has been overlooked and it is only by examining these patients over a period of years that it is possible to state that these children—and they form less than twenty per cent. of all patients injured—do not later develop mentally, emotionally and physically as they should on account of the effects of a prolonged mild increase of the intracranial pressure; an emotional instability is possibly the most common result, a mental retardation, and, as stated before, the great danger of epilepsy itself. This chronic cerebral edema persists in children less frequently than it does in adults, but its ultimate effects are more pronounced in children on account of the need for development of the cerebral nerve cells in all their activity; it is thus realized that the resulting functional impairment due to an increased intracranial pressure, if prolonged over a period of months and years, may produce a definite organic change of tissue—and then it is irreparable. The treatment should be the appropriate one at the time of the acute injury and not months and years later—when frequently only an improvement can be obtained and not a normal individual.

Case 189.—Old severe brain injury associated with a fracture of the base of the skull and with convulsive seizures; resulting mental and emotional impairment, mild secondary optic atrophy and an increased intracranial pressure. Operation advised but refused. No improvement.

No. 74.—Frank. Ten years. White. U. S.

Admitted March 22, 1914—15 months after injury. Polyclinic Hospital. Referred by Doctor E. W. Lawrence.

Discharged March 28, 1914—6 days after admission.

Family history negative. No nervous nor mental abnormality in the family of either parent; two other children well and strong.

Personal History.—First child; nine months' pregnancy; normal labor. No serious children's diseases; always well and strong, and up to the time of the cranial injury, the boy had done well in school.

Present Illness.—Fifteen months ago (December 6, 1912), patient was knocked down by a trolley-car while crossing the street; immediate loss of consciousness which continued for 8 hours; profuse bleeding from both ears; taken to a hospital where he remained 10 weeks under the expectant palliative treatment. During the first 7 days following the injury, patient had general convulsive seizures—as many as 8 in one day. He gradually improved, however, and yet his entire mental and emotional make-up became changed in that he is very quiet, sullen, refuses to speak, holds head down continuously, resists all attempts to move him and apparently does not notice anything; is dirty in his habits and he must be fed forcibly, as otherwise he will remain over 24 hours without eating; he must be dressed and taken care of as a child; walks about the house or sits by himself in silence, holding his head down and not interested in anything. This condition has persisted since his discharge from the hospital—neither becoming better nor worse; no complaints. No convulsions since his discharge from the hospital.

Examination upon admission (15 months after injury).—Temperature, 98.6°; pulse, 76; respiration, 20; blood-pressure, 110. Patient refuses to answer questions and pays no attention to anyone or his surroundings; holds head down and his eyes have a vacant stare; will not take candy offered to him nor express any emotion other than when an attempt is made to take his hand, he withdraws it forcibly (similar to the negativism of dementia præcox). No paralyses of the extremities nor sensory impairments can be elicited; no ocular paralysis. Unable to test the special senses on account of the patient's lack of coöperation. Otoscopic examination reveals both tympanic membranes thickened and retracted, and a small perforation in the lower posterior portion of the right tympanic membrane—the result of the former laceration at the time of the cranial injury. Pupils equal and react to light normally; no strabismus nor nystagmus elicited. Reflexes—patellar obtained with difficulty but apparently equal; no ankle clonus but a tendency to a left Babinski; abdominal reflexes equally depressed. Fundi—retinal veins enlarged and dilated in places; both optic disks rather pale from new tissue formation along the nasal margins and in the physiological cups which are rather shallow—right optic disk more than left; the nasal margins of both optic disks slightly blurred by edema. Lumbar puncture—clear cerebrospinal fluid under increased pressure (approximately 13 mm.); ten c.c. removed for examination: Wassermann test was negative and the cell count was only 4 per c.mm. X-ray (Doctor A. J. Quimby)—"no fracture of the skull found." This patient was repeatedly examined during his residence of 6 days in the hospital and the above signs were confirmed; as the patient resisted all attempts to study him with any degree of accuracy and refused to coöperate in any way, it was practically impossible to make a more thorough examination of his mental and emotional make-up.

Treatment.—The history of the cranial injury with the definite signs of an increased intracranial pressure persisting for a period of months and producing the mild secondary optic atrophy made it advisable to suggest the operation of cranial decompression in the hope that it would prevent a greater mental and emotional impairment and possibly obtain an improvement—the operative indication being the existing intracranial pressure which naturally should have been relieved at the time of the injury. The operation was refused. The examination at discharge was the same as recorded above.

Examination (September 20, 1916—45 months after injury and 30 months since the last examination).—The patient has remained in practically the same condition and possibly has gradually become a little worse in that he is even less observant than at the first examination; walks about less than before and must be dragged by the hand when moved; has never attempted to attack his brother or sister. No convulsive seizures. He resists all attempts to examine him as at the first examination and does not utter a sound. Pupils equal and react normally. Reflexes all depressed but otherwise negative. Fundi as at preceding examination.

Last Examination (November 2, 1918—71 months after injury).—No change in the condition of the patient has been observed; it is becoming so difficult, however, to take care of him at home that the parents are considering the advisability of sending him to an institution and my opinion is to that effect. All attempts to examine him are resisted as before and the physical examination remains practically the same.

Remarks.—In the absence of the history of severe intracranial injury and the persistence of an earlier and even present increased intracranial pressure, the diagnosis would naturally be one of early dementia præcox or even one of traumatic hysteria; in the presence of the history and the signs of an increased intracranial pressure as disclosed by the ophthalmoscopic and lumbar puncture findings, the more probable diagnosis is one of traumatic dementia following a severe cerebral injury and most probably due to multiple punctate hemorrhages throughout the cerebral cortex, producing the marked mental and emotional impairment with its acute onset and the formation of a persistent cerebral edema, which continues to be the cause of the present increased intracranial pressure. If an operation with drainage of the acute condition had been performed at the time of the injury (as soon as the initial shock had been overcome), it is very possible that the condition of this patient would not have become so severe as at present—at least the ophthalmoscopic findings, and it might have been hoped that the mental and emotional impairment would not have become so marked. It is possible that this severe intracranial injury has precipitated the condition of dementia præcox or at least a traumatic dementia with præcoid characteristics and this observation has been repeatedly made in other patients. The family history, however, of this patient is excellent.

Case 190.—Chronic severe brain injury occurring at one month of age associated with a linear fracture of the right vault and with a mild left spastic hemiplegia, mild mental retardation and occasional convulsive seizures; an increased intracranial pressure. Right subtemporal decompression; exploratory incision of the scalp overlying a possible depressed fracture of the vault. Marked improvement.

No. 970.—Mary. Three years. White. U. S.

Admitted April 2, 1918—2 years and 11 months after injury. Polyclinic Hospital. Referred by Doctor C. V. Niemeyer, Union Hill, N. J.

Operation April 13, 1918. Right subtemporal decompression; exploratory scalp incision.

Discharged April 26, 1918—13 days after operation.

Family history negative; four other children living and well.

Personal History.—Third child, full-term baby, normal labor, and no complications after birth; apparently a well child. When one month of age, child fell headlong from its carriage down an entire flight of stairs; loss of consciousness for fifteen minutes; no bleeding from nose, mouth or ears but right side of scalp contused and boggy; it seemed that the child was making an excellent recovery, when on the fourth day after the cranial injury a convulsive seizure of the left arm, left leg and left side of face occurred, but apparently no loss of consciousness (a Jacksonian epileptiform convulsion); the second convulsive seizure which became a general one occurred six weeks later and during the past 35 months, the child has had a convulsion always beginning in the left arm or left leg and becoming a general convulsive seizure every 6 to 8 weeks—the longest interval of freedom from convulsions; these attacks have lasted from one to three minutes and are accompanied by loss of sphincteric control and an occasional biting of the tongue. As the child developed, it was noted that the left arm a
leg were slightly more stiff and awkward than the right arm and th
leg; the child, however, walked with but a slight limp of the left side.
general development of the child was delayed in that she did not ho
head up and did not attempt to crawl or to walk as early as a normal
should; the speech was delayed and her present speech is slurred and
tinct; the mentality is retarded as well as the emotional reactions.

Examination upon admission (35 months after injury).—Temperature, 98.6°; pulse, 84; respiration, 26; blood-pressure, 114. Well-developed and nourished. Mentality slightly impaired in that the child is not so alert and is in a sort of confused hazy condition; rather irritable; unable to speak plainly and only in monosyllables; she walks with but a slight limp of the left leg—heel just touching the floor. Upon bimanual examination of the head, there is a possible depression of the right parietal bone; no tenderness. Hearing negative; otoscopic examination negative. Pupils equal and react to light normally. Reflexes—patellar exaggerated, left more than right; no ankle clonus but suggestive left Babinski; abdominal reflexes present and equal; reflexes of left arm more active than of right. Fundi—retinal veins enlarged; nasal margins of both optic disks blurred by edema—the other details being clear and distinct. Lumbar puncture—clear cerebrospinal fluid under increased pressure (16 mm.); Wassermann test negative and cell count was 4 cells per c.mm. X-ray (Doctor G. W. Welton)—"wide U-shaped fracture extending antero-posteriorly through middle portion of right parietal bone; no depressed fragments observed" (Fig. 212).

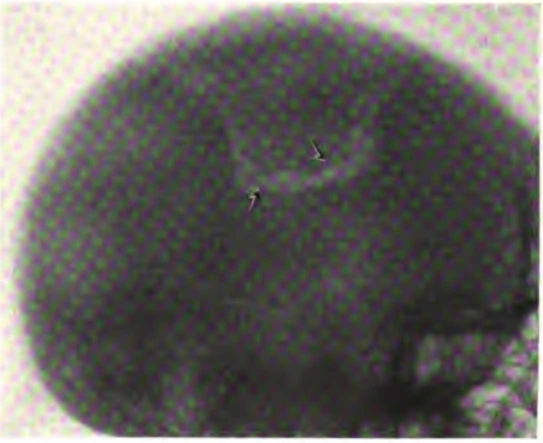


FIG. 212.—Irregular linear fracture of the right vault occurring in a child of one month of age; a resulting supracortical hemorrhage produced a left spastic hemiplegia with mental retardation and convulsive seizures. Marked improvement following a lowering of the increased intracranial pressure by means of a right subtemporal decompression and drainage.

Treatment.—On account of the increased intracranial pressure associated with convulsive seizures beginning in the left arm and in the left leg and with a mild spastic paralysis of the left side of the body with mental retardation, a right subtemporal decompression was advised and also an exploratory incision of the scalp overlying the right parietal bone—the area of a possible depressed fracture of the vault.

Operation (almost 3 years after injury).—Right subtemporal decompression; exploratory incision of the scalp. Usual vertical incision, bone removed and no complications. Dura thickened, whitish, tense and slightly bulging; upon incising the dura, a very "wet," edematous cortex tended to protrude but did not rupture owing to the escape of a large quantity of clear cerebrospinal fluid which at first spurted to a height of 2 inches; upon enlarging the dural opening, there was exposed a cloudy, whitish connective tissue formation about the vessels in the sulci—the organization of a former supracortical subarachnoid hemorrhage (and the cause of the partial blockage of the excretion of the cerebrospinal fluid and thus the formation of a mild external hydrocephalus—the "wet," edematous condition of the cortex). No gross cortical hemorrhage or laceration observed. Brain pulsated normally at the end of the operation. Usual closure with 2 drains of rubber tissue inserted. Duration, 30 minutes. A small curvilinear scalp incision was now made higher up over the right parietal bone and the possible depressed fracture; upon retracting the scalp, the large linear fracture, having a width of almost one-half inch, was found pulsating; no depression. It was considered better surgical judgment not to attempt an exploration of this area in the hope that the subtemporal decompression would be sufficient to obtain an excellent result. Usual closure. Duration, 65 minutes.

Post-operative Notes.—Uneventful convalescence in that incisions healed *per primam;* decompression area bulged, but pulsated normally; within one week, it was observed that the slight stiffness of the left arm and left leg had definitely lessened.

Examination at discharge (13 days after operation).—Temperature, 98.8°; pulse, 82; respiration, 24; blood-pressure, 112. Decompression area bulges slightly and pulsates normally; both scalp incisions have healed perfectly. Stiffness and awkwardness of left side of body definitely less than before the operation; child is possibly more interested in her surroundings. Pupils equal and of normal reaction to light. Reflexes: patellar—left more active than right; no ankle clonus and suggestive left Babinski persists; abdominal reflexes present and equal; reflexes of left arm less active than before the operation. Fundi—retinal veins slightly enlarged; edematous blurring of nasal margins of both optic disks less marked.

Treatment.—Continued as before the operation in that the diet is restricted by the avoidance of all meats, meat soups, tea and coffee; daily movement of the bowels; daily massage and exercises continued.

Last Examination (June 20, 1919—14 months after operation).—No convulsive seizures have occurred since the operation; a marked improvement has occurred in the lessening of the stiffness and awkwardness of the left arm and the left leg, so that now no impairment can be noted—child walks like a normal child; speech has improved, although there is still a slight hesitancy and slurring of words; both mentally and emotionally the improvement is marked. Reflexes: patellar—left slightly more active than right; no ankle clonus and no Babinski; reflexes of left arm slightly increased over those of right. Fundi—retinal veins of normal size; no edematous obscuration of the details of either optic disk. Decompression area depressed and pulsates normally; some new bone formation at the periphery slightly narrows the bony opening.

Remarks.—The cranial injury of this patient occurring 4 weeks after birth can be considered almost as being a birth injury and the resulting impairment of this patient, both mentally and physically and associated with convulsive seizures, is so characteristic of patients having an intracranial hemorrhage at the time of birth, that this patient may be classed wit
The absence of a depressed fracture of the right vault but the pres
an increased intracranial pressure associated with convulsive seizu
a mild spastic paralysis of the left side indicated the condition of
cortical hemorrhage of the right cerebral hemisphere and this
firmed at operation.

It is unfortunate that this child could not have been operated u time of the cranial injury and as soon as the signs of shock had disappeared, and in this manner not only would the mental and physical impairments have been avoided but the convulsive seizures have probably been prevented, for this early operation would have drained the blood in fluid form so that the secondary blockage of the cerebrospinal fluid would not have followed later to the extent that it did; although the improvement of this patient has been a most gratifying one following this late operation, yet it will be necessary to wait a period of years before it can be definitely stated that the recovery of the child, and especially from the convulsive seizures, is a permanent one and that this patient will become, or at least approximate, a normal person, both mentally and emotionally. The partial blockage of the cerebrospinal fluid naturally is a permanent one, but it is hoped that the operation of decompression will afford sufficient drainage to permit the amount of intradural cerebrospinal fluid to remain normal; in those patients where the blockage is even greater, then it is advisable to insert the linen strands for drainage subdurally just as in the cases of severe external hydrocephalus.

The rapid improvement of the physical condition of this patient and also of the mental retardation, including the difficulty of speech, is very impressive and merely confirms the belief that these impairments are merely functional to the extent that there is not a primary destruction of cerebral tissue, but that the impairments result from an increased intracranial pressure due to the partial blockage of the excretion of the cerebrospinal fluid and producing therefore a "wet," edematous condition of the cerebral cortex under varying degrees of pressure—a mild condition of external hydrocephalus; the therapeutic indication therefore is to lessen this increased intracranial pressure, either by the use of the thyroid extract in the very mild conditions by decreasing the amount of cerebrospinal fluid secreted, or by the operative and mechanical lowering of the higher degrees of increased intracranial pressure by means of the subtemporal decompression and drainage; naturally, the earlier this lessening of the increased intracranial pressure is possible, just so much greater is the improvement and the avoidance of later mental and physical impairments.

CASE 191.—Old severe brain injury associated with a depressed fracture of the left parietal vault and with signs of increased intracranial pressure; right hemiplegia and attacks of *petit mal.* Left subtemporal decompression and drainage. Improvement.

No. 38.—Albert. Twelve years. White. School. U. S.

Admitted October 20, 1913—5 years after injury. Polyclinic Hospital. Referred by Doctor O. S. Wightman.

Operation October 27, 1913. Left subtemporal decompression.

Discharged November 8, 1913—11 days after operation.

Family history negative; 2 older brothers and one younger sister well and strong; parents normal and of temperate habits; no miscarriages. Parents and relatives all right-handed.

Present History.—Patient is third child; full term baby, normal labor and no abnormalities observed after birth. Patient was considered a normal child until the cranial injury at 7 years of age. Five years ago, patient fell from a second-story window, striking upon the left side of his head; immediate loss of consciousness and bleeding from the left ear; taken to a hospital where the depressed area of the left parietal bone was elevated; patient remained unconscious for 5 days and unable to speak during the 2 weeks following the injury; definite weakness of entire right side of body so that at discharge, 4 weeks after injury, he was described as having "right spastic hemiplegia." Patient slightly improved during the first year after his discharge from the hospital, but no improvement has occurred during the past 4 years. No headache nor other complaints except the right spastic hemiplegia, a mental retardation and an emotional instability; during the past 2 months, patient has had several momentary losses of consciousness—staring blankly and dropping whatever was in his hand; no convulsions, however, at any time.

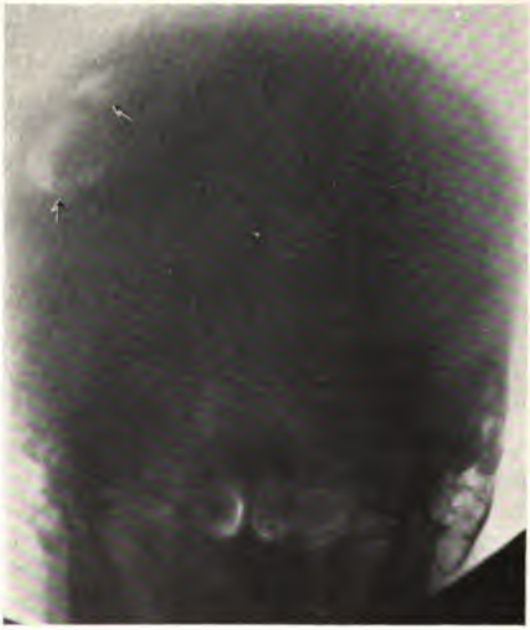


Fig. 213.—Posterior view of an irregular bony defect and new bone formation over the left vault in a boy of 12 years of age following a cranial injury five years before; the right spastic hemiplegia and epileptiform spells improved by lowering the increased intracranial pressure by means of a left subtemporal decompression and drainage.

Examination upon admission (5 years after injury).—Temperature, 98.6°; pulse, 84; respiration, 24; blood-pressure, 120. Well-developed and nourished. Over the left parietal region was a depressed area of bone as though some of the underlying bone had been removed at the former operation. Typical right spastic hemiplegia with the characteristic flexion of the right arm and position of the right leg due to the flexor contraction of the right foot, so that patient walked upon the toes of the right foot on account of the contraction of the Achilles tendon. Definite slurring of speech but no aphasia nor paraphasia. No sensory impairment. Pupils equal and react normally. Reflexes: patellar—right much more active than left; no ankle clonus but right Babinski; abdominal reflexes—right depressed. Fundi—retinal veins dilat
walls thickened with new tissue formation; nasal margins of
disks obscured and much new tissue formation about the margin
physiological cups. No lumbar puncture performed; Wassern
blood, however, was negative. X-ray (Doctor A. J. Quimby)—
bony defect of 2 cm. in diameter in left parietal area; slight d
its periphery; no linear fracture ascertained" (Fig. 213).

Treatment.—The presence of an increased intracranial pressure as revealed by the ophthalmoscopic examination, together with the definite right hemiplegia, made a left subtemporal decompression advisable in the hope that a lessening of this increased intracranial pressure would result in a lessened spasticity of the right arm and right leg, an improvement of the mentality made possible and the emotional instability improved.

Operation (5 years after injury).—Left subtemporal decompression: usual vertical incision, bone removed, and no complications; at upper portion of operative area, the bone was depressed and upon removing it, the underlying dura was found torn or incised at the former operation, and it was therefore thought advisable to remove only the lower portion of the overlying bone. The dura of the decompression area was now incised as usual, allowing clear cerebrospinal fluid to spurt under tension; upon enlarging the dural opening, the underlying cerebral cortex tended to protrude but did not rupture; bluish cystic formation beneath the arachnoid and above the Sylvian fissure; this was punctured, allowing straw-colored cerebrospinal fluid to escape. Owing to the escape of cerebrospinal fluid, the cortex became relaxed and pulsated normally. Usual closure with 2 drains of rubber tissue inserted. Duration, 40 minutes.

Post-operative Notes.—Uneventful operative recovery; cranial incision healed *per primam,* so that patient could be discharged 11 days after operation.

Examination (September 6, 1914—10 months after operation).—Patient has made a definite improvement in that the spasticity of the right arm and of the right leg has lessened and although he still limps, yet the right heel touches the floor and the lameness is not so marked; patient can use the right hand for simple duties (Fig. 214). Teacher in school says that he can now be taught more easily and is much more interested in things; also not so irritable. Mother says that no "fainting" spells have occurred since the operation. Decompression area slightly depressed and pulsates normally. Reflexes: patellar—right more active than left; no ankle clonus but right Babinski; right abdominal reflexes less active than left. Fundi—retinal veins slightly enlarged; nasal margins of optic disks no longer blurred by edema but new tissue formation naturally persists.

Examination (October 12, 1916—36 months after operation).—Patient has continued to improve both mentally and physically; the right spastic hemiplegia, however, is still present but much lessened in severity, so that patient now walks with but a slight limp; the condition of the right arm has improved but not so much as the right leg. Patient has advanced in school. No attacks of *petit mal* have occurred. Reflexes: patellar—right greater than left; no ankle clonus but right Babinski persists; right abdominal reflexes depressed. Fundi—retinal veins slightly larger than normal; optic disks clear but new tissue formation is present. Decompression area depressed and slight pulsation palpable.

Last Examination (November 6, 1918—60 months after operation).—Patient is practically the same as at preceding examination; the right hemiplegia is present but only in a modified degree compared with the condition before operation and the patient is mentally and emotionally more normal.

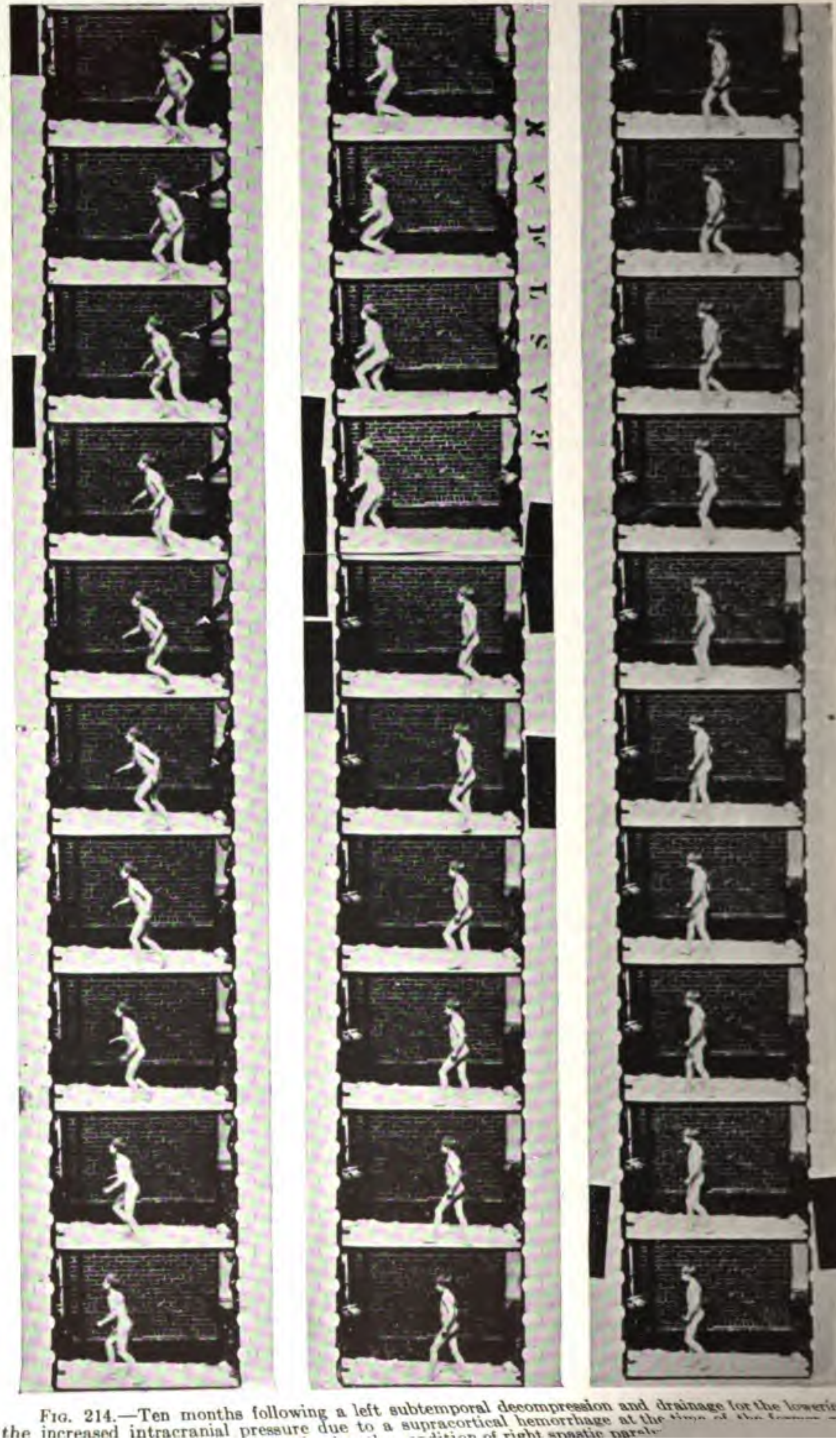


FIG. 214.—Ten months following a left subtemporal decompression and drainage for the lowering of the increased intracranial pressure due to a supracortical hemorrhage at the

No signs of *petit mal* attacks. Decompression area markedly depressed and its diameter is lessened due to new bone formation at the periphery. Reflexes: patellar—right more active than left; no ankle clonus but right Babinski; abdominal reflexes—right less than left. Fundi as at preceding examination.

Remarks.—The condition of this patient resembles so closely and in such detail the condition of those children having cerebral spastic paralysis due to an intracranial hemorrhage at the time of birth—due usually to a difficult labor; in this case, however, we have a child who was normal until the age of 7 years, when the cranial injury occurred; it would seem that a subtemporal decompression and drainage should have been performed at the time of the injury and not merely the elevation of the depressed area of bone performed; undoubtedly, there has been some destruction of the cortical nerve cells supplying the right arm and the right leg and naturally a complete recovery therefore cannot be expected. It is encouraging, however, that during a period of 5 years following the decompression operation, no spells of *petit mal* character have appeared again and it is to be hoped that the lessening of the increased intracranial pressure will prevent this frightful complication from occurring. It will be very interesting to watch this patient over a number of years—not so much what his present condition is, but what his future condition will be 15 to 20 years from now, when he will begin to assume the duties of manhood.

Case 192.—Chronic severe brain injury associated with a depressed fracture of the vault of the skull and with a resulting hemiplegia and mental retardation; an increase of the intracranial pressure. Left subtemporal decompression and then a removal of the depressed area of bone. Marked improvement.

No. 791.—John. Ten years. White. School. U. S.

Admitted February 20, 1917—3 years after injury. Polyclinic Hospital. Referred by Doctor John A. Wyeth.

Operations.—First, February 28, 1917: left subtemporal decompression. Second, March 7, 1917—7 days after the first operation: removal of depressed area of vault.

Discharged March 16, 1917—9 days after second operation.

Family history negative.

Personal History.—Third child, full term, normal labor, and no complications. Child was considered a normal one in every way until he was seven years of age (3 years ago), when he fell from a ladder, striking upon the left side of his head; immediate loss of consciousness and was taken in the ambulance to St. Vincent's Hospital, where he remained three months; there was a paralysis of the right arm and right leg observed upon the day following the injury when a right-sided convulsion occurred. As the condition of paralysis slightly improved and as no other convulsive seizures occurred, no operation was performed, and at the end of three months the patient was discharged from the hospital. Since then, there has remained a definite spastic paralysis of the right arm and of the right leg and the patient has been unable to advance in school more than one class during the past 3 years; frequent severe headaches have been complained of by the

[illegible] no convulsions since [illegible]er injury).—Temperature, [illegible], 114. Patient is rather dull [illegible]sted in his surroundings and very irritable upon being chided. [illegible]miplegic type—the right leg being [illegible]; the lower right side of the face is [illegible] right facial paralysis). Upon local [illegible] definite depressed fracture of the left [illegible] one-half inch wide and easily one-fourth [illegible] horizontally about one and a half inches to [illegible] not tender. Hearing negative, apparently, [illegible] upon the patient's answers as he seems rather [illegible] however, negative. Pupils equal and react [illegible]—patellar active, right much more than left; [illegible] ankle clonus; right Babinski, Oppenheim and [illegible] reflexes—right depressed and very difficult to [illegible] veins enlarged with thickened walls; both optic [illegible] new tissue formation about the nasal margins and [illegible]—the left possibly more than the right optic disk; optic disks blurred by edema. Lumbar puncture— under increased pressure (14 mm.). X-ray (Doctor [illegible] evidence of a displaced section of the left vault."

[illegible] history of the cranial injury with the resulting right [illegible] and the present findings of a definite increase of the [illegible] and the depressed fracture of the left vault (not [illegible] roentgenogram) with the right spastic hemiplegia persisting [illegible] mental retardation and emotional instability—these facts [illegible] of left subtemporal decompression advisable and then [illegible] removal of the depressed area of the left parietal bone, if [illegible] by the bimanual examination.

[illegible] (3 years after injury).—Left subtemporal decompres[illegible] vertical incision, bone removed, and no complications. Dura [illegible] dense, thickened and vascular; upon incising it, much clear [illegible] fluid welled out, and upon enlarging the dural opening the [illegible] edematous cortex tended to protrude but did not rupture, owing [illegible] escape of the cerebrospinal fluid. At the upper portion of the [illegible] field and above the Sylvian fissure was exposed a film of a fibrous [illegible] formation the result of a former supracortical hemorrhage which [illegible] extended from above downward over the parietal area of the cortex. [illegible] the cortical vessels in the sulci in this same area was a whitish indura[illegible] the organization of the former free blood about the vessels in the [illegible] The overlying arachnoid was punctured with a needle in several [illegible] allowing a straw-colored fluid to escape and the cystic formation to [illegible] At the end of the operation the cortex pulsated normally. Usual [illegible] with 2 drains of rubber tissue inserted. Duration, 35 minutes.

[illegible] Notes.—No complications; operative incision healed *per*

hile the operative area itself bulges beyond the flush of scalp normally. An attempt was now made to remove the depressed vault.

d Operation (7 days after first operation).—Removal of depressed vault: curvilinear incision over the depressed area of the left parie- one of 3 inches in length; upon retraction of the scalp, it was found at there was a longitudinal depressed fracture of the left parietal bone about 2 inches in length, one-half inch in width and almost one-half inch in depth. It was possible to insert the rongeurs at the edge of the bony depression, so that the bony opening could be enlarged and the depressed fragment of bone was removed. The underlying dura was not torn and it was considered advisable and better surgical judgment not to open it as the left subtemporal decompression had lowered the increased intracranial pressure and it was now too late to remove the supracortical hemorrhage, since it had become organized and only a lowering of the intracranial pressure and the removal of the bony local compression were considered necessary. Usual closure with 2 drains of rubber tissue inserted down the dura. Duration, 25 minutes.

Post-operative Notes.—Uneventful operative recovery; incision healed *per primam;* within 5 days, patient no longer complained of the dull headache and said his head "felt lighter." Decompression area bulged but pulsated normally.

Examination at discharge (9 days after second operation).—Temperature, 98.6°; pulse, 82; respiration, 24; blood-pressure, 116. Patient appears somewhat brighter in that he is more interested in his surroundings; no complaints other than a soreness of left side of head. Decompression area bulges beyond the flush of scalp; pulsation normal. Right arm and right leg possibly not so spastic as before the operation; the patient walks with less of a limp; is using the right hand possibly better. Pupils negative. Reflexes: patellar—right more active than left; exhaustible right patellar clonus and right ankle clonus; right Gordon, Oppenheim and Babinski reflexes persist; right abdominal reflex less active than left. Fundi—retinal veins enlarged; no change from former fundal examination unless the nasal margins of the optic disks are not so obscured by edema as before the operation. Another röntgenogram taken, showing the bony defect of removing the depressed area of bone (posterior view) (Fig. 215).

Treatment.—Parents were advised the usual hygienic rules, a non-proteid diet and the avoidance of school until the next fall.

Examination (April 20, 1918—13 months after operation).—Child has made a marked improvement during the past year in that the spasticity of the right arm and leg has so lessened that the patient can walk much more freely and can use the right arm less awkwardly than before the operation; the right side of face is still slightly weak and lags in its movements. The mentality has also improved and he is able to study and enjoy it; no longer complains of headaches and has had only "two attacks of temper" during the past 6 months. The decompression area extends beyond the flush of the surrounding scalp but pulsates normally. Pupils equal and react to light normally. Reflexes: patellar—right more active than left, patient and the emotional irritability has increased; no convulsions since the day following the injury.

Examination upon admission (3 years after injury).—Temperature, 98.6°; pulse, 78; respiration, 24; blood-pressure, 114. Patient is rather dull and non-observant; apparently not interested in his surroundings and answers questions in a slow hazy manner; very irritable upon being chided. Marked spastic paralysis of the right hemiplegic type—the right leg being possibly affected more than the right arm; the lower right side of the face is also impaired (the cortical type of right facial paralysis). Upon local bimanual examination, there is a definite depressed fracture of the left parietal bone almost 3 inches long, one-half inch wide and easily one-fourth of an inch in depth, extending horizontally about one and a half inches to the left of the longitudinal sinus; not tender. Hearing negative, apparently, although it is difficult to rely upon the patient's answers as he seems rather confused; otoscopic examination, however, negative. Pupils equal and react normally to light. Reflexes—patellar active, right much more than left; right patellar and right ankle clonus; right Babinski, Oppenheim and Gordon reflexes; abdominal reflexes—right depressed and very difficult to elicit. Pupils—retinal veins enlarged with thickened walls; both optic disks slightly pale from new tissue formation about the nasal margins and in the physiological cups—the left possibly more than the right optic disk; nasal margins of both optic disks blurred by edema. Lumbar puncture—clear cerebrospinal fluid under increased pressure (14 mm.). X-ray (Doctor W. H. Stewart)—"no evidence of a displaced section of the left vault."

Treatment.—The history of the cranial injury with the resulting right spastic hemiplegia and the present findings of a definite increase of the intracranial pressure and the depressed fracture of the left vault (not confirmed by the röntgenogram) with the right spastic hemiplegia persisting and a marked mental retardation and emotional instability—these facts made the operation of left subtemporal decompression advisable and then the elevation or removal of the depressed area of the left parietal bone, if found as indicated by the bimanual examination.

First Operation (3 years after injury).—Left subtemporal decompression: usual vertical incision, bone removed, and no complications. Dura moderately tense, thickened and vascular; upon incising it, much clear cerebrospinal fluid welled out, and upon enlarging the dural opening the underlying edematous cortex tended to protrude but did not rupture, owing to the rapid escape of the cerebrospinal fluid. At the upper portion of the operative field and above the Sylvian fissure was exposed a film of a fibrous cystic formation—the result of a former supracortical hemorrhage which had extended from above downward over the parietal area of the cortex. About the cortical vessels in the sulci in this same area was a whitish i
tion—the organization of the former free blood about the vessels
sulci. The overlying arachnoid was punctured with a needle in
places, allowing a straw-colored fluid to escape and the cystic form
collapse. At the end of the operation the cortex pulsated normally
closure with 2 drains of rubber tissue inserted. Duration, 35 minu

Post-operative Notes.—No complications; operative incision h *primam,* while the operative area itself bulges beyond the flush of scalp but pulsates normally. An attempt was now made to remove the depressed area of the vault.

Second Operation (7 days after first operation).—Removal of depressed area of vault: curvilinear incision over the depressed area of the left parietal bone of 3 inches in length; upon retraction of the scalp, it was found that there was a longitudinal depressed fracture of the left parietal bone about 2 inches in length, one-half inch in width and almost one-half inch in depth. It was possible to insert the rongeurs at the edge of the bony depression, so that the bony opening could be enlarged and the depressed fragment of bone was removed. The underlying dura was not torn and it was considered advisable and better surgical judgment not to open it as the left subtemporal decompression had lowered the increased intracranial pressure and it was now too late to remove the supracortical hemorrhage, since it had become organized and only a lowering of the intracranial pressure and the removal of the bony local compression were considered necessary. Usual closure with 2 drains of rubber tissue inserted down the dura. Duration, 25 minutes.

Post-operative Notes.—Uneventful operative recovery; incision healed *per primam;* within 5 days, patient no longer complained of the dull headache and said his head "felt lighter." Decompression area bulged but pulsated normally.

Examination at discharge (9 days after second operation).—Temperature, 98.6° ; pulse, 82; respiration, 24; blood-pressure, 116. Patient appears somewhat brighter in that he is more interested in his surroundings; no complaints other than a soreness of left side of head. Decompression area bulges beyond the flush of scalp; pulsation normal. Right arm and right leg possibly not so spastic as before the operation; the patient walks with less of a limp; is using the right hand possibly better. Pupils negative. Reflexes: patellar—right more active than left; exhaustible right patellar clonus and right ankle clonus; right Gordon, Oppenheim and Babinski reflexes persist; right abdominal reflex less active than left. Fundi—retinal veins enlarged; no change from former fundal examination unless the nasal margins of the optic disks are not so obscured by edema as before the operation. Another röntgenogram taken, showing the bony defect of removing the depressed area of bone (posterior view) (Fig. 215).

Treatment.—Parents were advised the usual hygienic rules, a nonproteid diet and the avoidance of school until the next fall.

Examination (April 20, 1918—13 months after operation).—Child has made a marked improvement during the past year in that the spasticity of the right arm and leg has so lessened that the patient can walk much more freely and can use the right arm less awkwardly than before the operation; the right side of face is still slightly weak and lags in its movements. The mentality has also improved and he is able to study and enjoy it; no longer complains of headaches and has had only "two attacks of temper" during the past 6 months. The decompression area extends beyond the flush of the surrounding scalp but pulsates normally. Pupils equal and react to light normally. Reflexes: patellar—right more active than left;

no right patellar clonus but an exhaustible right ankle clonus; right Babinski still persists but neither an Oppenheim or Gordon reflex can be elicited; right abdominal reflexes depressed. Fundi—retinal veins slightly larger than normally; both optic disks rather pale from connective tissue formation as before the operation, but there is no edematous obscuration of the nasal margins. Visual acuity: 16/20 in the left eye and 18/20 in the right eye—the result of a mild secondary optic atrophy; no limitation of the visual fields ascertained.

Last Examination (May 14, 1919—26 months after operation).—The improvement has continued during the past year so that the right side of the body can be used with less awkwardness than before and the mentality of the patient has become much brighter, more alert and his emotional reactions are more stable; right facial paresis persists but is hardly noticeable (Fig. 221); he has advanced with his class in school, although he is still 2 years behind his regular class. No complaints of headache or convulsive seizures. Decompression area bulges beyond the flush of the scalp. Reflexes: patellar—right greater than left; slight exhaustible right ankle clonus persists and right Babinski; right abdominal reflexes less active than left. Fundi—the same as at preceding examination. Visual acuity remains practically the same as at the preceding examination one year ago.

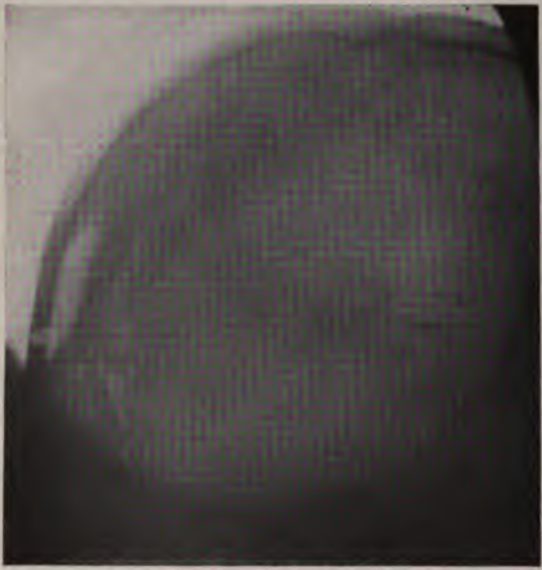


FIG. 215.—Irregular bony defect of left vault following the removal of depressed bone in a boy having the condition of right spastic hemiplegia with mental retardation. Marked improvement following the operation and the lowering of the increased intracranial pressure by means of a left subtemporal decompression and drainage.

Treatment.—The massage, daily exercises and his mental training are advised to be continued as before, together with the routine hygienic measures.

Remarks.—The similarity of these patients having depressed fractures of the vault of the skull and with a resulting increase of the intracranial pressure to those patients having a spastic paralysis with mental retardation and emotional instability as the result of an intracranial hemorrhage at the time of birth with a resulting increased intracranial pressure—this similarity is very great indeed and in reality the condition is the same, except that in the patients having an increased intracranial pressure resulting from an intracranial hemorrhage at birth, the results of the condition are usually more pronounced because the increased intracranial pressure occurred during the earliest stage of mental and emotional development, and therefore not only is the retardation greater but it is a much more serious and permanent one; the normal development of the cortical cells is prevented and therefore the mental and physical impairment is more pronounced than that which follows an intracranial hemorrhage of similar degree occurring in a child who has already developed mentally and physically as is normal for the age at which the intracranial hemorrhage occurred. Merely because a depressed fracture of the vault is not present in only a very small percentage of children having an intracranial hemorrhage at the time of birth—this is no reason to consider these patients as being essentially different pathologically and clinically from the ones showing external evidence of cranial injury, such as a depressed fracture of the skull; the important factor to ascertain is the presence or not of an increased intracranial pressure due to intracranial hemorrhage or persistent cerebral edema, and if found to be present, then an operation of decompression and drainage should be performed to lower it—whether there is a depressed fracture of the skull or not; in this manner, the mental and physical impairment and retardation cannot only be improved but—more important—be prevented if the operation is performed early. Naturally, all depressed fractures of the vault should be elevated or removed for fear of future complications such as epilepsy; the danger of convulsive seizures occurring later in this patient was very great indeed and they are still to be feared even with the apparent success of the operations performed.

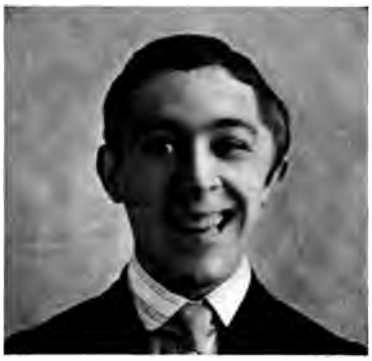


FIG. 216.—The area of the left decompression bulges so markedly that there can be no doubt as to the increased intracranial pressure in this patient and the necessity of lessening it in order to obtain an improvement. The right facial weakness is much less than before the operation.

On account of the increased intracranial pressure, it was better surgical judgment to lower this pressure first by means of the subtemporal decompression performed on the same side as the depressed area of the vault, and then it was possible to remove the depressed area of the vault safely and with no risk to the underlying cerebral cortex; to have opened the cerebral cortex underlying the depressed area of the vault in this patient and a decompression not having been previously performed, would have been of great danger to the patient as the more highly developed areas of this underlying cortex would have become damaged by their protrusion owing to the high intradural pressure and, in this manner, the end-result could not have been so successful as when the decompression is performed first rather than after the local operation upon the depressed area of the vault.

The supracortical pathology as ascertained at this operation is very typical of the condition as found in practically all of the patients having a spastic paralysis resulting from an intracranial hemorrhage at the time of birth; that is, a supracortical fibrous and cystic formation occurs as the result of the non-absorption of the free subdural and subarachnoid hemorrhagic clot and the connective tissue formation as the result of the organization of the free blood about the vessels in the sulci; in this latter manner, the stomata of exit for the normal excretion of the cerebrospinal fluid through the walls of the cortical veins in the sulci are partially blocked and therefore a mild "wet," edematous condition of the brain results—a mild secondary external hydrocephalus.

Case 194.—Chronic severe brain injury associated with a fracture of the vault of the skull; apparently an excellent recovery until the later development of an internal hydrocephalus and the signs of a tumor of the right half of the cerebellum—most probably a tuberculoma. Right subtemporal decompression and permanent ventricular drainage. Marked improvement.

No. 1081.—Elmer. Nine years. White. School. U. S.

Admitted July 22, 1919—9 months after cranial injury. Audubon Hospital. Referred by Doctor E. B. McCready, Pittsburgh.

Operation July 30, 1919. Right subtemporal decompression and ventricular drainage.

Discharged August 6, 1919—7 days after operation.

Family History.—Father died from "pneumonia" three years ago; mother living and well; three brothers living and well.

Personal History.—Third child, full term, normal labor. Usual childhood diseases and apparently in the best of health during the two years preceding the cranial injury. Nine months ago (November 6, 1918), while the patient was playing in the street, he was knocked down by an automobile; immediate loss of consciousness with profuse bleeding from the left ear; he was taken to a hospital—remaining unconscious for 2 days and finally recovered so that he could be discharged at the end of 2 weeks; (an X-ray picture taken at that time disclosed a "linear fracture of the left half of the occipital bone and extending forward toward the left auditory canal"). Patient apparently made an excellent recovery and within one month he appeared to be perfectly well. Two weeks later, however (7½ months ago), patient began to complain of dull frontal and occipital headaches, early fatigue and a general weakness; within 2 weeks, he began to stagger in walking, tending to fall toward the right side and also backward; attacks of vomiting now occurred irrespective of eating. One month later, it was noticed that the child was unable to see as clearly as formerly so that his school teacher had his seat placed at the head of the class; the headaches continued, the difficulty of standing and of walking became greater until 3 months ago, the child was unable to walk or to stand alone owing to his staggering and falling to the right and backward. The impairment of vision increased until he could not distinguish fingers or large objects with his left eye and only with difficulty with his right eye. His condition had been diagnosed as an irreparable brain injury due to the cranial injury at the time of the accident—a destruction of cerebral tissue and therefore a hopeless condition.

Examination upon admission (9 months after injury).—Temperature, 98.6°; pulse, 82; respiration, 26. Well-developed and nourished; rather drowsy and confused mentally. Patient complains of severe frontal headache and inability to see. Head larger than the average, although within physiological limits; upon palpation, a definite linear fracture of the left half of the occipital bone extends forward into the left mastoid area; a possible depression of the bone just posterior to the left mastoid area. Hearing of left ear markedly impaired; bone conduction greater than air conduction; otoscopic examination discloses a small perforation in the lower posterior half of the left tympanic membrane which is whitish, thickened and retracted. Patient is blind in the left eye and can only distinguish light with it, whereas with the right eye he can count fingers and distinguish large objects but with great difficulty. Coarse nystagmoid twitches toward the right but none toward the left. No weakness of the ocular muscles. Definite weakness of the right side of face in its entire distribution (the peripheral type of facial paralysis). No impairment of hearing of the right ear. No impairment of sensation of the right side of the face. The right arm and the right leg are slightly weaker than the left arm and left leg. Distinct intention tremor of both hands, especially the right hand in the pointing tests. No definite speech defect or slurring of words; no impairment of swallowing. Typical Romberg in that the patient always falls to the right and backward; in attempting to walk, he staggers to the right dragging the right leg. Pupils: slightly enlarged but equal; sluggish reaction to light. Reflexes—patellar very much exaggerated but equal; no ankle clonus; bilateral Babinski; abdominal reflexes absent. Fundi—retinal veins dilated, tortuous and buried in places in edematous tissue; double "choked disks" of 5 diopters of swelling and much new tissue formation so that it is assuming a whitish appearance of a progressive secondary optic atrophy; this latter condition is more advanced in the left eye. X-ray (Doctor A. J. Quimby)—"wide linear fracture of the left vault posteriorly extending downward to left mastoid area" (Fig. 217). Lumbar puncture—clear cerebrospinal fluid under high pressure (38 mm.); Wassermann test negative and cell count was 14 cells per c.mm. (Only 2 c.c. of the cerebrospinal fluid were removed and very carefully, for fear that medullary complications might be induced.) Tuberculin skin test negative.

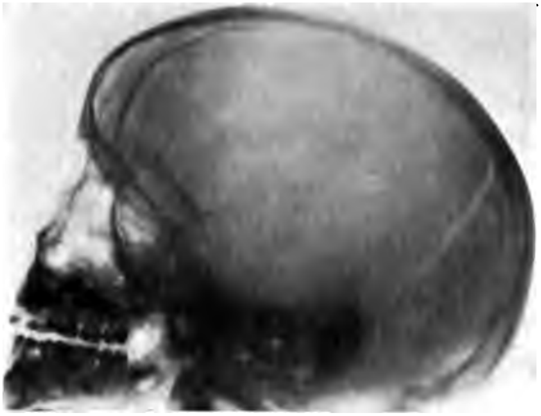


FIG. 217.—Extensive linear fracture of the posterior portion of the left vault in a boy developing later an internal hydrocephalus due most probably to a subtentorial tuberculoma. Marked improvement following a right subtemporal decompression and permanent ventricular drainage. The pressure atrophy of the vault and the convolutional pressure markings are more noticeable in the frontal area.

Treatment.—The presence of the signs of very high intracranial pressure sufficient to produce a secondary optic atrophy would indicate a severe degree of internal hydrocephalus due to a subtentorial or posterior basal lesion or to a very large cerebral tumor, and since the secondary optic atrophy had advanced more in the left eye, the greater possibility of a tumor of the left cerebral hemisphere. The clinical signs, however, would indicate a lesion of the right half of the cerebellum—the homolateral weakness of the right arm and right leg, the intention tremor more marked in the right hand, the coarse nystagmus to the right and the right facial paresis of peripheral type (this last sign being more indicative of a right cerebellopontine angle tumor, but the absence of an impairment of hearing of the right ear or of sensation of the right side of the face would tend to exclude this localization). The history of the cranial injury and the resulting fracture of the posterior vault of the skull is very interesting; possibly this injury lessened the resistance of the underlying cerebellar tissue by contusion, etc., so that the formation of a tuberculoma in them was facilitated—just as injuries to the joints of the body render them more susceptible to disease and especially to tuberculosis; the injury, however, may have occurred after the intracranial lesion had been in existence for some time and the injury itself merely precipitated its progress. It was considered advisable to perform first a right subtemporal decompression and at the same time establish permanent ventricular drainage in the belief that the lesion was a tuberculous one and if the effects of its presence, and particularly the blockage of the ventricles, could be obviated, then it would not be necessary (nor is it advisable) to attempt a removal of the growth itself; besides, the risk of this operative procedure of subtemporal decompression and ventricular drainage is a slight one.

Operation (9 months after cranial injury).—Right subtemporal decompression and ventricular drainage: usual vertical incision, bone removed, and no complications; the bone itself was so thin, as a result of a prolonged increase of the intracranial pressure, that it was of a thickness of not more than a sheet of paper—so thin that it could be depressed by digital compression similar to a "ping-pong" depression. Dura under high tension, bulging into bony opening and similar to a drum in tightness; its vessels had been entirely compressed and obliterated by the prolonged tension; upon making a small opening in it, no cerebrospinal fluid escaped, and it was considered better judgment to attempt a ventricular puncture and removal of ventricular cerebrospinal fluid before enlarging the dural opening for fear of the underlying cerebral cortex being ruptured by the high tension. Ventricle puncture needle was inserted to a depth of 2 cm. when clear cerebrospinal fluid spurted through the needle to a height of 5 inches; a large quantity of it escaped, permitting the dural tension to lessen and the cortex to pulsate almost normally. Six linen strands were inserted into the ventricle and permitted to extend through the temporal muscle and fascia outward into the subcutaneous tissues of the scalp in a stellate manner, and as a means of permanent drainage of the ventricles, just as in conditions of internal hydrocephalus in children. The dural opening was now enlarged as in the usual decompression operation and as the cortex pulsated normally owing to the escape of so much ventricular cerebrospinal fluid, it was possible to close the operative incision in the usual manner with 2 drains of rubber tissue inserted subdurally. Duration, 50 minutes.

Post-operative Notes.—Uneventful operative recovery in that the incision healed *per primam* and the general condition of the patient so improved that he could be discharged from the hospital upon the eighth day post-operative; at that time the headaches had ceased, his mental condition had cleared, and the "choked disks" of 5 diopters had lessened to a condition of papilledema of only 1 diopter of swelling; the vision of the right eye had definitely improved and he was able to stagger unsupported for a distance of 7 or 8 steps. The operative area bulged slightly beyond the flush of the surrounding scalp and pulsated normally.

Last Report—December 6, 1919 (5 months after operation).—Letter from district nurse states that, "marked improvement continued during the first three months after the operation, but he is at a stand-still for the last six weeks; the operative incision bulges tensely; no complaints, however."

Remarks.—In spite of the history that this boy was perfectly well at the time of the cranial injury nine months ago, it is very difficult to conceive that the intracranial pressure had not been increased for a longer period than nine months on account of the marked pressure atrophy of the bones of the vault so that they were only of the thickness of paper, together with the advanced degree of secondary optic atrophy. The history, however, is negative before the cranial injury—no headaches, no dizzy spells nor attacks of vomiting and until the accident occurred, he had been considered an unusually healthy boy. Although the cranial injury may have been the primary cause of the increased intracranial pressure and the possible tumor formation, yet it is more probable that the condition itself would have developed irrespective of the cranial injury, and that the cranial injury was merely an incident in the clinical history of this patient or, at most, it had precipitated the acute ventricular blockage and the more rapid progress of the growth of the tumor. It must be remembered, however, that the pathology in this patient may not be that of a tumor formation, but that the ventricular dilatation is the result of adhesions occurring in the subtentorial fossa following the organization of a subtentorial hemorrhage which had occurred there at the time of the cranial injury and that the signs of right cerebellar disease were due to trauma at the time of the cranial injury of the right half of the cerebellum; the absence of these latter signs, however, is significant until the patient had entirely recovered from the effects of the cranial injury for a period of one month following it.

The more advanced stage of the secondary optic atrophy being present in the left eye, and especially a greater visual impairment of this eye, may have resulted in part from a direct damage to the left occipital lobe lying beneath the site of fracture of the left half of the occipital bone; this is very much doubted, however, because there were no signs of visual impairment of either eye until 2 months following the cranial injury, and a direct trauma to the left occipital lobe would have produced immediately a right homonymous hemianopsia—and this was at least not ascertained.

The positive tuberculin test is significant, especially in the absence of a pulmonary focus, or one to be found elsewhere in the body; the increased cell count of the cerebrospinal fluid, and especially the presence of lymphocytes, were very suggestive; also the fact that tuberculomata, and particularly of the base and subtentorial fossa, are the most common forms of intracranial tumor occurring in children under 12 years of age and particularly cerebellar tumors—these facts would tend to indicate that this tumor—if a tumor in reality is present—is one of tuberculous character. There were no signs of tuberculous nodules upon the pia-arachnoid in the sulci and about the supracortical vessels as disclosed at operation; these findings are rather common in the presence of tuberculomata at the base.

Naturally, a longer period of time must elapse following the operation before it can be stated with any degree of certainty that this patient has been permanently improved. It is most unfortunate, however, that the high intracranial pressure could not have been lowered at a much earlier date than at least 9 months following its inception—the impairment of vision could have been thus prevented and a more hopeful prognosis assured—at least temporarily. A further report of this patient will be made later in a series of cases of brain tumors.

Case 195.—Chronic severe brain injury followed by an increasing intracranial pressure with resulting secondary optic atrophy, left hemiplegia and mental impairment. Right subtemporal decompression and exploration disclosing a large subcortical tumor of the right occipital lobe—a tuberculoma. Death; post-mortem examination.

No. 1083.—David. Eight years. White. School. U. S.

Admitted August 12, 1919—15 months after cranial injury. Audubon Hospital. Referred by Doctor G. F. Boehme.

Operation August 20, 1919. Right subtemporal decompression and exploration.

Died August 21, 1919—16 hours after operation.

Family history negative; two brothers and one sister younger than the patient and all perfectly well; no miscarriages for mother.

Personal History.—First child, full term, instrumental delivery (low forceps); apparently normal after birth and except for the usual diseases of childhood, the patient was perfectly well in every way until the cranial injury. Fifteen months ago, while playing upon a shed, the patient fell headlong to the ground, striking the right side of the back of his head against a stone; immediate loss of consciousness; no bleeding from nose, mouth or ears. Patient was taken home and remained unconscious for 16 hours and then semiconscious during the following week; gradually recovered so that at the end of 6 weeks, patient was considered well enough to return to school; no abnormalities were observed by his teacher or relatives. Ten months ago (5 months after cranial injury), child began to complain of severe frontal headache on an average of twice a week and lasting for a period of 24 hours. Three months later (7 months ago), severe vomiting spells occurred following the beginning of the weekly headaches and with no relation to the meals—in fact, the patient refused to eat anything after the headaches began. Four months later (3 months ago), it was
for the first time that the child was unable to see as well as form
this visual impairment has rapidly progressed during the past three
during which time it has been noticed that the left arm and left
become definitely weaker than the right arm and right leg and th
side of the face exhibits the cortical type of left facial paralysi
forehead muscles not being involved). Patient has not been al

alone during the past 6 weeks and he always falls toward the left when not supported. Mentality has become very much dulled while the headaches have become continuous. No convulsive seizures at any time.

Examination upon admission (15 months after cranial injury and 10 months after the first complaints).—Temperature, 98.6°; pulse, 84; respiration, 26. Well-developed and nourished. Rather drowsy and not interested in his surroundings. Head rather large and rachitic in type; veins of the scalp enlarged and numerous; venules of upper eyelids dilated. Definite left hemiplegia but not complete; unable to walk alone and he drags the left leg; unable to hold objects in his left hand. No sensory impairment elicited and no astereognosis ascertained. Left facial paralysis of the cortical type. No nystagmus or ocular paralyses elicited. No impairment of speech. No intention tremor of either hand. Hearing negative; otoscopic examination negative. Vision markedly impaired in that he cannot distinguish objects with either eye—only the difference between light and darkness (no hemianopsia could therefore be ascertained at this late stage). Pupils equal, slightly enlarged, but react to light normally. Reflexes: patellar very much exaggerated, left more than right; no ankle clonus but left Babinski well defined and only a suggestive right Babinski at times; abdominal reflexes—left absent, right depressed; deep reflexes of left arm increased. Fundi—retinal veins dilated, tortuous and buried in edematous tissue in places; bilateral "choked disks" of 6 diopters with beginning connective tissue formation so that a whitish appearance is presented. Lumbar puncture—clear cerebrospinal fluid under high pressure (40 mm.); Wassermann test negative and the cell count was 14 cells per c.mm. and all were lymphocytes. X-ray (Doctor A. J. Quimby)—"there are indentures in the inner plate of the skull, corresponding to cerebral convolutions and indicating an increased intracranial pressure; the sella turcica is above normal in size" (Fig. 218). Tuberculin skin test negative.

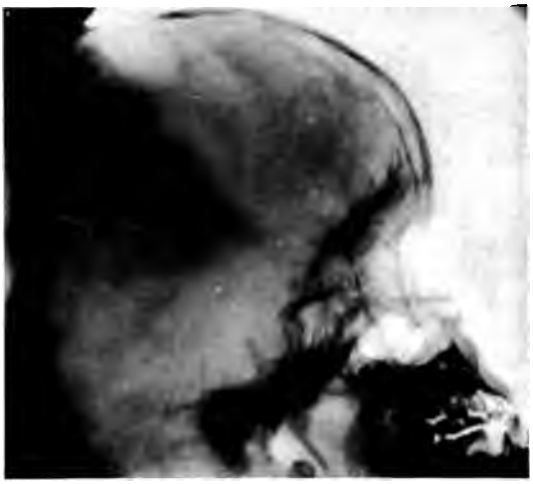


FIG. 218.—Convolutional markings of pressure atrophy of the inner table of the vault due to a prolonged increase of the intracranial pressure in a patient developing a large tuberculoma of the right occipital lobe following an injury of the overlying vault of the skull.

Treatment.—The high intracranial pressure sufficient to produce a bilateral choking of the disks with a resulting secondary optic atrophy should have been lowered months ago, whether a definite diagnosis of the intracranial lesion could have been made or not—the therapeutic indication is the same and that is to lower the increased intracranial pressure and therefore prevent the visual impairment at least and then later, it may be possible to locate and to diagnose the intracranial lesion itself and if possible it can then be removed—and the patient be not blind. The left hemiplegia in the absence of the definite signs of subtentorial and cerebellar disease indicated a lesion, and most probably a large tumor, of the right cerebral hemisphere and therefore a right subtemporal decompression and exploration was advised.

Operation (15 months after cranial injury).—Right subtemporal decompression and exploration: usual vertical incision, bone removed, and no complications; the thickness of the bone was not more than that of a sheet of paper and due to prolonged pressure atrophy. Dura pale, non-vascular and under high tension; in the hope that the ventricles might be dilated and therefore the cerebral tension lowered by means of a ventricular puncture, so that little or no damage would occur to the underlying cortex upon enlarging the dural opening, a small dural incision was made and a ventricle puncture needle inserted to a depth of 3 cm., when a small amount of clear cerebrospinal fluid escaped and then ceased; another puncture about one inch posteriorly also permitted a small amount of clear cerebrospinal fluid to escape under tension, but the right lateral ventricle could not be considered as being dilated. It was therefore obligatory to enlarge the dural opening without first having been able to lower markedly the high intracranial pressure and it was feared that the underlying cerebral cortex of the temporal lobe would be damaged. Upon enlarging widely the dural opening, a small amount of clear cerebrospinal fluid escaped and the underlying cerebral cortex tended to protrude through the dural opening. Using the blunt ventricular puncture needle to explore the right cerebral hemisphere, a tumor mass, the size of an orange, was located subcortically in the right occipital lobe and near the base; as there were small whitish nodules scattered about the supracortical vessels in the sulci in the pia-arachnoid, the co was considered as being one of tuberculous character and the tum probably a large tuberculoma, so that no attempt was made to remov fear of precipitating an acute tuberculous meningitis. The presenc tumor in this location explained the collapse of the right ventricle large size of the tumor produced the high intracranial pressure the absence of ventricular blockage. Usual closure with 2 drains of

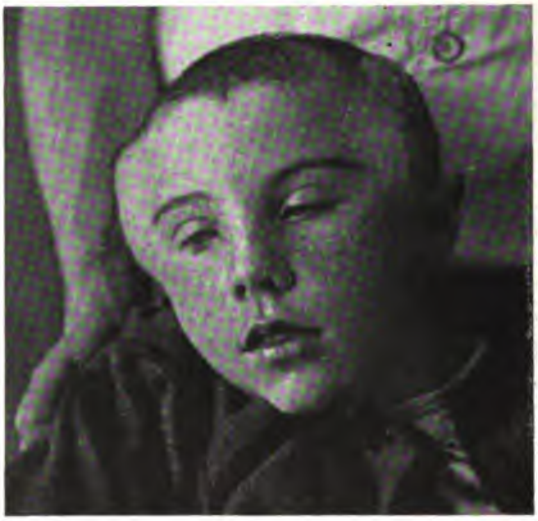


Fig. 219.—Extreme intracranial pressure in a patient having a large tuberculoma of the right occipital lobe and causing a marked protrusion of the right decompression area and the subsequent death of the patient.

tissue inserted; it was necessary, in order to obtain an accurate closure of the temporal muscle and fascia, to sacrifice a small portion of the right temporal lobe which had been herniated through the decompression opening by the high cerebral tension and this was accomplished with no complications. Duration, one hour.

Post-operative Notes.—A very acute cerebral edema occurred as a result of the operative trauma; the decompression area bulged tensely and the child did not regain complete consciousness (Fig. 219). Within eight hours, the temperature ascended to 106.4°, the pulse- and respiration-rates to 148 and 38, respectively, and the child died 16 hours after the operation.

Post-mortem Examination.—No fracture of the skull ascertained. Cerebral pressure had been so high that the right temporo-sphenoidal lobe was herniated into the decompression opening. No gross hemorrhage. Tumor mass, the size of an orange, in the right occipital lobe—2 cm. beneath the cortex and not attached to the base of the skull (Fig. 220). Throughout the pia-arachnoid were numerous tuberculous nodules. Right ventricle compressed and almost obliterated by the tumor mass; no blockage of the third ventricle. Pathological report of tumor (Doctor Jeffries)—"a tuberculoma."

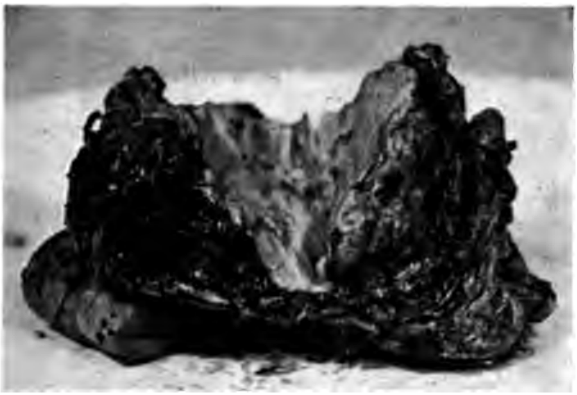


Fig. 220.—A tuberculoma of the right occipital lobe of the size of a small orange—2½ inches in diameter. A secondary optic atrophy of advanced degree prevented the presence of an earlier left homonymous hemianopsia from being ascertained.

Remarks.—The operative findings of a large subcortical tumor of the right occipital lobe, together with the whitish nodules in the pia-arachnoid about the supracortical vessels in the sulci and in the presence of an increased cell count of the cerebrospinal fluid and especially of lymphocytes, pointed to this tumor as being a tuberculoma—in spite of the negative tuberculin skin test, a negative test being possibly more common in this type of tuberculosis than in any other form; this opinion was confirmed by the report of the pathologist.

If this patient could have been examined earlier before such a marked degree of secondary optic atrophy had occurred, it is very probable that a left homonymous hemianopsia could have been demonstrated due to an impairment of the visual cells and their tracts in the right occipital lobe; it would have been then a comparatively simple matter to locate the intracranial lesion, although, as has been stated repeatedly in this book, it is not of so much importance to the patient to locate accurately the lesion as it is to offset its pressure effects—spare the vision, lessen the mental impairment and then if the lesion is a removable one surgically, to do so after a decompression has first been performed, so that the increased intracranial pressure has been sufficiently lowered to permit a safe removal of the lesion with as little damage as possible to the surrounding cerebral tissues.

It is rare for an increased intracranial pressure to reach such a height as to produce "choked disks" of 5 diopters plus and, if prolonged, its resulting secondary optic atrophy, unless the ventricles are blocked and thus producing an internal hydrocephalus or unless the tumor is a very large supratentorial one; naturally, the blockage of the ventricles may be due to a small tumor situated in the posterior basal areas and subtentorially, and also very frequently due to a meningeal exudate and resulting adhesions; it is rare for an external hydrocephalus due to blockage of the stomata of exit of the cerebrospinal fluid into the supracortical veins and sinuses to produce a sufficiently high intracranial pressure as to cause the condition of double "choked disks" and the resulting secondary optic atrophy.

These case-histories have been reported in detail in the hope that they will be of assistance in the diagnosis and treatment of similar conditions occurring in new-born babies, in children and in adults; the appropriate treatment—whether the expectant palliative or the operative—instituted at the time of the acute condition and not delayed in its chronic form until years later, in the hope that the patient will "outgrow" it. An attempt has been made to recognize the common mistakes in the diagnosis by routine post-mortem examinations for the benefit of future patients and thus avoid the burying of our mistakes with the patient.

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